EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	6	"5176145"	USPAT	OR	ON	2007/04/27 12:26
L2	2	"20030216366"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 12:19
L3	3353	desipramine	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 12:27
L4	281	"hot flushes"	USPAT	OR	ON	2007/04/27 12:27
L5	556	"hot flashes"	USPAT	OR	ON	2007/04/27 12:27
L6	790	L4 or L5	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 12:27
L7	17	L3 and L6	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 12:43
L8	574	"vasomotor symptoms"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 12:44
L9	34	L3 and L8	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:03

EAST Search History

L10	184	atipamezole	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:23
L11	83	L3 and L10	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:08
L12	5	L10 and L8	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:10
L13	0	L10 and L6	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:12
L14	0	"91186602"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:12
L15	5	"9118602"	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:12
L16	5838	clonidine	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:23

EAST Search History

L17	53	L10 and L16	US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2007/04/27 13:24
S1	26	"4229449"	USPAT	OR	ON	2007/04/27 11:56
S2	7	"4310524"	USPAT	OR	ON	2007/04/04 11:49
S3	40	"4535186"	USPAT	OR	ON	2007/04/04 11:53
S4	2	"4826844"	USPAT	OR	ON	2007/04/04 11:58
S5	5	"5502047"	USPAT	OR	ON	2007/04/04 11:58
S6	5	"5502047"	USPAT	OR	ON	2007/04/26 12:29
S7	41	"4535186"	USPAT	OR	ON	2007/04/26 13:11
S8	1269	desipramine	USPAT	OR	ON	2007/04/26 13:11
S9	281	"hot flushes"	USPAT	OR	ON	2007/04/26 13:11
S10	2	S8 and S9	USPAT	OR	ON	2007/04/26 13:13
S11	556	"hot flashes"	USPAT	OR	ON	2007/04/26 13:13
S12	15	S8 and S11	USPAT	OR	ON	2007/04/26 13:37
S13	1045	"vasomotor"	USPAT	OR	ON	2007/04/26 13:38
S14	39	S8 and S13	USPAT	OR	ON	2007/04/26 13:38

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                  with preparation role
 NEWS  4  DEC 18  CA/CAplus patent kind codes updated
 NEWS  5  DEC 18  MARPAT to CA/CAplus accession number crossover limit increased
                  to 50,000
 NEWS  6  DEC 18  MEDLINE updated in preparation for 2007 reload
 NEWS  7  DEC 27  CA/CAplus enhanced with more pre-1907 records
 NEWS  8  JAN 08  CHEMLIST enhanced with New Zealand Inventory of Chemicals
 NEWS  9  JAN 16  CA/CAplus Company Name Thesaurus enhanced and reloaded
 NEWS 10  JAN 16  IPC version 2007.01 thesaurus available on STN
 NEWS 11  JAN 16  WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data
 NEWS 12  JAN 22  CA/CAplus updated with revised CAS roles
 NEWS 13  JAN 22  CA/CAplus enhanced with patent applications from India
 NEWS 14  JAN 29  PHAR reloaded with new search and display fields
 NEWS 15  JAN 29  CAS Registry Number crossover limit increased to 300,000 in
                  multiple databases
 NEWS 16  FEB 15  PATDPASPC enhanced with Drug Approval numbers
 NEWS 17  FEB 15  RUSSIAPAT enhanced with pre-1994 records
 NEWS 18  FEB 23  KOREAPAT enhanced with IPC 8 features and functionality
 NEWS 19  FEB 26  MEDLINE reloaded with enhancements
 NEWS 20  FEB 26  EMBASE enhanced with Clinical Trial Number field
 NEWS 21  FEB 26  TOXCENTER enhanced with reloaded MEDLINE
 NEWS 22  FEB 26  IFICDB/IFIPAT/IFIUDB reloaded with enhancements
 NEWS 23  FEB 26  CAS Registry Number crossover limit increased from 10,000
                  to 300,000 in multiple databases
 NEWS 24  MAR 15  WPIDS/WPIX enhanced with new FRAGHITSTR display format
 NEWS 25  MAR 16  CASREACT coverage extended
 NEWS 26  MAR 20  MARPAT now updated daily
 NEWS 27  MAR 22  LWPI reloaded
 NEWS 28  MAR 30  RDISCLOSURE reloaded with enhancements
 NEWS 29  MAR 30  INPADOCDB will replace INPADOC on STN
 NEWS 30  APR 02  JICST-EPLUS removed from database clusters and STN

 NEWS EXPRESS  NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

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=> s desipramine
L1        35180 DESIPRAMINE

=> s norepinephrine reuptake
L2         2505 NOREPINEPHRINE REUPTAKE

=> s vasomotor
L3        34332 VASOMOTOR

=> s L1 and L3
L4           51 L1 AND L3

=> dup rem L4
PROCESSING COMPLETED FOR L4
L5            41 DUP REM L4 (10 DUPLICATES REMOVED)

=> s L5 and L2
L6            4 L5 AND L2

=> s L5 and (AY<2003 or PY<2003 or PRY<2003)
'2003' NOT A VALID FIELD CODE
'2003' NOT A VALID FIELD CODE
   2 FILES SEARCHED...
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'2003' NOT A VALID FIELD CODE
'2003' NOT A VALID FIELD CODE
L7           35 L5 AND (AY<2003 OR PY<2003 OR PRY<2003)

=> s L7 and atipamezole
L8            2 L7 AND ATIPAMEZOLE

=> d 1-4 L6 ibib abs

L6   ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2005:369266  CAPLUS
DOCUMENT NUMBER:          142:404276
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TITLE:                   Method using adrenergic α2B antagonists, alone
                         or in combination with norepinephrine
                         reuptake inhibitors or dual
                         norepinephrine/serotonin reuptake inhibitors for
                         treating vasomotor symptoms
INVENTOR(S):             Deecher, Darlene Coleman; Beyer, Chad Edward;
                         Leventhal, Liza
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 48 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  3
PATENT INFORMATION:

     PATENT NO.          KIND   DATE         APPLICATION NO.        DATE
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     WO 2005037260        A2    20050428     WO 2004-US33754        20041013
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             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     US 2004152710        A1    20040805     US 2003-685812         20031014
     WO 2004035058        A1    20040429     WO 2003-US32759        20031015
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE,
             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
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     US 2005130987        A1    20050616     US 2004-962897         20041012
     AU 2004281750        A1    20050428     AU 2004-281750         20041013
     CA 2539757           A1    20050428     CA 2004-2539757        20041013
     BR 2004015280        A     20061219     BR 2004-15280          20041013
PRIORITY APPLN. INFO.:                       US 2003-510897P     P  20031014
                                             US 2003-685812      A  20031014
                                             WO 2003-US32759     A  20031015
                                             US 2004-962897      A  20041012
                                             US 2002-418591P     P  20021015
                                             WO 2004-US33754     W  20041013
AB   The invention discloses selective adrenergic α2B antagonists alone,
     selective adrenergic α2B antagonists in combination with
     norepinephrine reuptake inhibitors (NRI) (as a single
     compound or as a combination of two or more compds.), or selective
     adrenergicα2B antagonists in combination with dual
     norepinephrine reuptake inhibitors/serotonin reuptake
     inhibitors (NRI/SRI) (as a single compound or as a combination of two or
     more compds.) and methods of their use in the treatment of
     vasomotor symptoms.

L6   ANSWER 2 OF 4  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2004:354797  CAPLUS
DOCUMENT NUMBER:         140:350606
```

TITLE: Use of norepinephrine reuptake modulators for preventing and treating vasomotor symptoms

INVENTOR(S): Deecher, Darlene Coleman; Merchenthaler, Istvan Joseph; Leventhal, Liza; Sipe, Kimberly Jean; O'Connor, Lawrence Thomas

PATENT ASSIGNEE(S): Wyeth, John, and Brother Ltd., USA

SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

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     PATENT NO.          KIND   DATE       APPLICATION NO.         DATE
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     WO 2004035058        A1    20040429   WO 2003-US32759         20031015
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             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
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     US 2004152710        A1    20040805   US 2003-685812          20031014
     CA 2502032           A1    20040429   CA 2003-2502032         20031015
     AU 2003282861        A1    20040504   AU 2003-282861          20031015
     EP 1551413           A1    20050713   EP 2003-774853          20031015
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     BR 2003015355        A     20050823   BR 2003-15355           20031015
     CN 1705484           A     20051207   CN 2003-80101535        20031015
     JP 2006506372        T     20060223   JP 2004-545349          20031015
     US 2005130987        A1    20050616   US 2004-962897          20041012
     AU 2004281750        A1    20050428   AU 2004-281750          20041013
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     WO 2005037260        A2    20050428   WO 2004-US33754         20041013
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             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
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             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
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             EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
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             SN, TD, TG
     BR 2004015280        A     20061219   BR 2004-15280           20041013
PRIORITY APPLN. INFO.:                     US 2002-418591P      P  20021015
                                           US 2003-685812       A  20031014
                                           US 2003-510897P      P  20031014
                                           WO 2003-US32759      W  20031015
                                           US 2004-962897       A  20041012
                                           WO 2004-US33754      W  20041013
```

AB The invention discloses the use of compds. and composition of compds. that modulate norepinephrine levels for the prevention and treatment of vasomotor symptoms, such as hot flush, caused by, inter alia, thermoregulatory dysfunctions. Compds. of the invention include e.g. desipramine.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS

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                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6   ANSWER 3 OF 4  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2004:354778  CAPLUS
DOCUMENT NUMBER:         140:350603
TITLE:                   A method of treating vasomotor symptoms
                         using a compound having norepinephrine
                         reuptake inhibitor activity and 5-HT2a
                         antagonistic activity
INVENTOR(S):             Deecher, Darlene Coleman; Merchenthaler, Istvan Joseph
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 34 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.          KIND    DATE          APPLICATION NO.        DATE
     ---------------     ----    --------      --------------------   --------
     WO 2004035036        A1     20040429      WO 2003-US32554        20031015
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             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
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     US 2004180879        A1     20040916      US 2003-685974         20031014
     CA 2502027           A1     20040429      CA 2003-2502027        20031015
     AU 2003282830        A1     20040504      AU 2003-282830         20031015
     EP 1551380           A1     20050713      EP 2003-774828         20031015
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
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     BR 2003015346        A      20050823      BR 2003-15346          20031015
     CN 1705475           A      20051207      CN 2003-80101558       20031015
     JP 2006516023        T      20060615      JP 2004-545270         20031015
PRIORITY APPLN. INFO.:                         US 2002-418516P     P  20021015
                                               US 2003-685974      A  20031014
                                               WO 2003-US32554     W  20031015
AB   The invention discloses the use of compds. and compns. of compds. that
     modulate norepinephrine levels for the treatment of vasomotor
     symptoms, e.g. thermoregulatory disorders.  The invention also discloses
     the use of compds. and compns. of compds. having norepinephrine
     reuptake inhibitor (NRI) activity alone or norepinephrine
     reuptake inhibitor and serotonin reuptake inhibitor (NRI/SRI) dual
     activity in combination with 5-HT2a receptor antagonist activity.
REFERENCE COUNT:         6     THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6   ANSWER 4 OF 4  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2004:354777  CAPLUS
DOCUMENT NUMBER:         140:350602
TITLE:                   Use of norepinephrine reuptake
                         modulators for preventing and treating
                         vasomotor symptoms
INVENTOR(S):             Deecher, Darlene Coleman; Merchenthaler, Istvan
                         Joseph; Leventhal, Liza; Sipe, Kimberly Jean;
                         O'Connor, Lawrence Thomas
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 58 pp.
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                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  3
PATENT INFORMATION:

     PATENT NO.             KIND   DATE        APPLICATION NO.         DATE
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     WO 2004035035           A1    20040429    WO 2003-US32760         20031015
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             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
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     US 2004143008           A1    20040722    US 2003-684777          20031014
     CA 2502021              A1    20040429    CA 2003-2502021         20031015
     AU 2003282862           A1    20040504    AU 2003-282862          20031015
     EP 1551379              A1    20050713    EP 2003-774854          20031015
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     BR 2003015314           A     20050816    BR 2003-15314           20031015
     CN 1705474              A     20051207    CN 2003-80101522        20031015
     JP 2006516243           T     20060629    JP 2004-545350          20031015
PRIORITY APPLN. INFO.:                         US 2002-418591P      P  20021015
                                               US 2003-684777       A  20031014
                                               WO 2003-US32760      W  20031015
AB   The invention discloses the use of compds. and compns. of compds. that
     modulate norepinephrine levels for the prevention and treatment of
     vasomotor symptoms, e.g. hot flush, caused by, inter alia,
     thermoregulatory dysfunctions.  Compds. of the invention include e.g.
     venlafaxine.
REFERENCE COUNT:         7     THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> s hot flushes
L9         3352 HOT FLUSHES

=> s L5 and L9
L10           0 L5 AND L9

=> d 1-10 L5 ibib abs

L5   ANSWER 1 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2006314319  EMBASE
TITLE:               Use of antidepressants during perimenopause.
AUTHOR:              Kim D.R.; Joffe H.
CORPORATE SOURCE:    D.R. Kim, Department of Psychiatry, Hospital of the
                     University of Pennsylvania, 3535 Market Street,
                     Philadelphia, PA 19104, United States.
                     drkim@mail.med.upenn.edu
SOURCE:              Women's Health, (2006) Vol. 2, No. 4, pp. 627-637. .
                     Refs: 44
                     ISSN: 1745-5057  E-ISSN: 1745-5065
COUNTRY:             United Kingdom
DOCUMENT TYPE:       Journal; General Review
FILE SEGMENT:        003     Endocrinology
                     010     Obstetrics and Gynecology
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                    032     Psychiatry
                    037     Drug Literature Index
                    038     Adverse Reactions Titles
LANGUAGE:           English
SUMMARY LANGUAGE:   English
ENTRY DATE:         Entered STN: 19 Jul 2006
                    Last Updated on STN: 19 Jul 2006
AB   This article reviews the literature on the use of antidepressants for
     symptoms associated with perimenopause.  In some perimenopausal women,
     mood instability, insomnia and vasomotor symptoms cause
     significant distress.  Studies of antidepressants for perimenopausal
     symptoms are summarized, with a focus on perimenopausal depression and
     vasomotor symptoms.  Antidepressants should be considered as an
     alternative to hormone therapy for perimenopausal depression and hot
     flashes, especially when hormone therapy is contraindicated, or as an
     augmentation strategy for women who are only partially responsive to
     hormone therapy. .COPYRGT.2006 Future Medicine Ltd.

L5   ANSWER 2 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2005:369266  CAPLUS
DOCUMENT NUMBER:         142:404276
TITLE:                   Method using adrenergic α2B antagonists, alone
                         or in combination with norepinephrine reuptake
                         inhibitors or dual norepinephrine/serotonin reuptake
                         inhibitors for treating vasomotor symptoms
INVENTOR(S):             Deecher, Darlene Coleman; Beyer, Chad Edward;
                         Leventhal, Liza
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 48 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  3
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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     WO 2005037260       A2     20050428    WO 2004-US33754        20041013
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
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             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     US 2004152710       A1     20040805    US 2003-685812         20031014
     WO 2004035058       A1     20040429    WO 2003-US32759        20031015
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
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             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
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             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2005130987       A1     20050616    US 2004-962897         20041012
     AU 2004281750       A1     20050428    AU 2004-281750         20041013
     CA 2539757          A1     20050428    CA 2004-2539757        20041013
     BR 2004015280        A     20061219    BR 2004-15280          20041013
PRIORITY APPLN. INFO.:                      US 2003-510897P      P  20031014
                                            US 2003-685812       A  20031014
                                            WO 2003-US32759      A  20031015
                                            US 2004-962897       A  20041012
                                            US 2002-418591P      P  20021015
                                            WO 2004-US33754      W  20041013
AB   The invention discloses selective adrenergic α2B antagonists alone,
     selective adrenergic α2B antagonists in combination with
     norepinephrine reuptake inhibitors (NRI) (as a single compound or as a
     combination of two or more compds.), or selective adrenergicα2B
     antagonists in combination with dual norepinephrine reuptake
     inhibitors/serotonin reuptake inhibitors (NRI/SRI) (as a single compound or
     as a combination of two or more compds.) and methods of their use in the
     treatment of vasomotor symptoms.

L5   ANSWER 3 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2005:369229  CAPLUS
DOCUMENT NUMBER:         142:430298
TITLE:                   Preparation of alkanol and cycloalkanol-amines, in
                         particular piperazine derivatives, as monoamine
                         reuptake modulators
INVENTOR(S):             Trybulski, Eugene John; Mahaney, Paige Erin; Krim,
                         Lori Danielle; Moore, William Jay; Sabatucci, Joseph
                         Peter
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 355 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.          KIND   DATE          APPLICATION NO.        DATE
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     WO 2005037207        A2    20050428    WO 2004-US33730          20041013
     WO 2005037207        A3    20060921
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             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
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             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     US 2005171115        A1    20050804    US 2004-962880           20041012
     AU 2004281745        A1    20050428    AU 2004-281745           20041013
     CA 2539755           A1    20050428    CA 2004-2539755          20041013
     EP 1673059           A2    20060628    EP 2004-794957           20041013
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             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
     BR 2004015292        A     20061219    BR 2004-15292            20041013
PRIORITY APPLN. INFO.:                      US 2003-510942P      P  20031014
                                            US 2004-561422P      P  20040412
                                            US 2004-569884P      P  20040511
                                            US 2004-962880       A  20041012
                                            WO 2004-US33730      W  20041013
OTHER SOURCE(S):         MARPAT 142:430298
GI
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* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

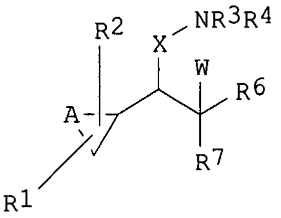
AB Title compds. I [W = H, OH and derivs.; R1, R2 = independently H, OH, alkyl, alkoxy, halo, CF3, alkanoyloxy, or together form methylene or methylenedioxy; R5 = H, alkyl; R6, R7 = independently cyclo/alkyl; or R6CR7 = (un)substituted 4-8 membered ring optionally fused with a (un)substituted carbocyclic or aromatic ring; R8 = H, alkyl; B = (CH2)m; A = (CH2)n; n = 1-3; m = 0-2] and their pharmaceutically acceptable salts, are prepared and disclosed as monoamine reuptake modulators. Thus, e.g., II•2HCl was prepared from (3-chlorophenyl)(4-hydroxytetrahydro-2H-pyran-4-yl)acetic acid (preparation given) and tert-Bu piperazine-1-carboxylate. In norepinephrine (NE) uptake assays, selected compds. (@ 1 μM), in the presence of 1 μM desipramine, inhibited the uptake of [3H]NE in 71-93%. I as monoamine reuptake inhibitors should prove useful in the treatment of conditions related to monoamine reuptake such as, but not limited to, vasomotor symptoms, major depressive disorders, and stress.

L5 ANSWER 4 OF 41 CAPLUS COPYRIGHT 2007 ACS on STN

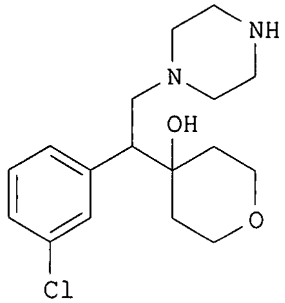
ACCESSION NUMBER: 2005:1132915 CAPLUS
DOCUMENT NUMBER: 143:387067
TITLE: Preparation of amino- and cycloamino-cycloalkanols, including piperazinyl derivatives, as monoamine reuptake modulators
INVENTOR(S): Mahaney, Paige Erin; Webb, Michael Byron; Gavrin, Lori Krim; Trybulski, Eugene John
PATENT ASSIGNEE(S): Wyeth, John, and Brother Ltd., USA
SOURCE: U.S. Pat. Appl. Publ., 157 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

	PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
	US 2005234058	A1	20051020	US 2004-962899		20041012
PRIORITY APPLN. INFO.:				US 2003-511001P	P	20031014
				US 2004-561459P	P	20040412
				US 2004-570072P	P	20040511

OTHER SOURCE(S): MARPAT 143:387067
GI



I



II

AB Title compds. I [A = (un)substituted Ph, naphthyl, thiophenyl, pyridinyl, etc.; W = H, OH and derivs.; R1, R2 = independently H, OH, alkyl, alkoxy, halo, CF3, alkanoyloxy, etc.; R3 = H [sic]; R4 = (un)substituted benzyl, naphthylmethyl, phenylalkyl, heteroarylmethyl, cycloalkylmethyl with 1 or more C atoms optionally replaced with N, O, S, etc.; R6, R7 = independently cyclo/alkyl, or R6CR7 = 4 to 8 carbocyclyl ring with 1 or more C atoms optionally replaced with N, O, S; R6CR7 = a ring with 4 to 8 C's fused onto a cycloalkyl ring of 4 to 6 C atoms; X = (CH2)n; n = 1-3] and their pharmaceutically acceptable salts, are prepared and disclosed as monoamine reuptake modulators. Examples are also presented in which NR3R4 = piperidin-1-yl, morpholin-4-yl, pyrrolidin-1-yl, piperazin-1-yl, etc. Thus, e.g., II•2HCl was prepared from (3-chlorophenyl)(4-hydroxytetrahydro-2H-pyran-4-yl)acetic acid (preparation given) and tert-Bu piperazine-1-carboxylate. In norepinephrine (NE) uptake assays, selected compds. (@ 1 μM), in the presence of 1 μM desipramine, inhibited the uptake of [3H]NE in 65-93%. I as monoamine reuptake inhibitors should prove useful in the treatment of conditions related to monoamine reuptake such as, but not limited to, vasomotor symptoms, major depressive disorders, and stress.

L5 ANSWER 5 OF 41 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005219761 EMBASE
TITLE: [Clinical uses of methadone].
USOS CLINICOS DE LA METADONA.
AUTHOR: Morillas Sendin P.; Fernandez Del Valle D.; Diz Gomez J.C.; Canovas Martinez L.; Carregal Rano A.
CORPORATE SOURCE: P. Morillas Sendin, Servicio de Anestesiologia, Reanimacion y Terapeutica del Dolor, Complexe Hospitalario de Ourense, C/ Ramon Puga, 54, 32005 Ourense, Spain. palomamorillas@wanadoo.es
SOURCE: Actualizaciones en Anestesiologia y Reanimacion, (2005) Vol. 15, No. 1, pp. 18-27. .
Refs: 45
ISSN: 1132-0095 CODEN: AANREK
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 024 Anesthesiology
030 Pharmacology

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                    036     Health Policy, Economics and Management
                    037     Drug Literature Index
                    038     Adverse Reactions Titles
LANGUAGE:           Spanish
SUMMARY LANGUAGE:   English; Spanish
ENTRY DATE:         Entered STN: 2 Jun 2005
                    Last Updated on STN: 2 Jun 2005
AB   Methadone is a synthetic opioid that is primarily a μ-opioid agonist,
     but also a NMDA-antagonist.  It is highly liposoluble and has an average
     life in blood of approximately 23 hours, although it can vary from 13 to
     54 hours.  It is an optical isomer, where 1-methadone is the primarily
     responsible factor for the analgesic effect.  Methadone has successfully
     been used for many years for the treatment of dependence on opioid drugs.
     However, it has been less used as an analgesic, in spite of its useful
     pharmacologic profile.  Bearing this in mind, we have reviewed the main
     therapeutic uses of methadone, and its effectiveness in treating both
     acute and chronic pain.  Methadone is an attractive alternative for the
     treating cancer and neuropathic pain, because of its good oral
     bioavailability, its long average life at low daily doses and its low
     cost.  Intravenous, intrathecal and epidural viae have shown to be
     effective and safe for the treatment of postoperative pain.  Copyright
     .COPYRGT. 2005 Aran Ediciones, S.  L.

L5   ANSWER 6 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2004:354797  CAPLUS
DOCUMENT NUMBER:          140:350606
TITLE:                    Use of norepinephrine reuptake modulators for
                          preventing and treating vasomotor symptoms
INVENTOR(S):              Deecher, Darlene Coleman; Merchenthaler, Istvan
                          Joseph; Leventhal, Liza; Sipe, Kimberly Jean;
                          O'Connor, Lawrence Thomas
PATENT ASSIGNEE(S):       Wyeth, John, and Brother Ltd., USA
SOURCE:                   PCT Int. Appl., 65 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   3
PATENT INFORMATION:

     PATENT NO.          KIND   DATE          APPLICATION NO.        DATE
     ---------------     ----   --------      --------------------   --------
     WO 2004035058        A1    20040429      WO 2003-US32759        20031015
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             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2004152710        A1    20040805      US 2003-685812         20031014
     CA 2502032           A1    20040429      CA 2003-2502032        20031015
     AU 2003282861        A1    20040504      AU 2003-282861         20031015
     EP 1551413           A1    20050713      EP 2003-774853         20031015
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             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     BR 2003015355        A     20050823      BR 2003-15355          20031015
     CN 1705484           A     20051207      CN 2003-80101535       20031015
     JP 2006506372        T     20060223      JP 2004-545349         20031015
     US 2005130987        A1    20050616      US 2004-962897         20041012
     AU 2004281750        A1    20050428      AU 2004-281750         20041013
     CA 2539757           A1    20050428      CA 2004-2539757        20041013
     WO 2005037260         A2     20050428    WO 2004-US33754        20041013
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             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     BR 2004015280         A      20061219    BR 2004-15280          20041013
PRIORITY APPLN. INFO.:                        US 2002-418591P     P  20021015
                                              US 2003-685812      A  20031014
                                              US 2003-510897P     P  20031014
                                              WO 2003-US32759     W  20031015
                                              US 2004-962897      A  20041012
                                              WO 2004-US33754     W  20041013
AB   The invention discloses the use of compds. and composition of compds. that
     modulate norepinephrine levels for the prevention and treatment of
     vasomotor symptoms, such as hot flush, caused by, inter alia,
     thermoregulatory dysfunctions.  Compds. of the invention include e.g.
     desipramine.
REFERENCE COUNT:         5     THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 7 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2004:354778  CAPLUS
DOCUMENT NUMBER:         140:350603
TITLE:                   A method of treating vasomotor symptoms
                         using a compound having norepinephrine reuptake
                         inhibitor activity and 5-HT2a antagonistic activity
INVENTOR(S):             Deecher, Darlene Coleman; Merchenthaler, Istvan Joseph
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 34 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.            KIND   DATE        APPLICATION NO.        DATE
     ---------------       ----   --------    --------------------   --------
     WO 2004035036         A1     20040429    WO 2003-US32554        20031015
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             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2004180879         A1     20040916    US 2003-685974         20031014
     CA 2502027            A1     20040429    CA 2003-2502027        20031015
     AU 2003282830         A1     20040504    AU 2003-282830         20031015
     EP 1551380            A1     20050713    EP 2003-774828         20031015
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     BR 2003015346         A      20050823    BR 2003-15346          20031015
     CN 1705475            A      20051207    CN 2003-80101558       20031015
     JP 2006516023         T      20060615    JP 2004-545270         20031015
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PRIORITY APPLN. INFO.:                    US 2002-418516P     P  20021015
                                          US 2003-685974      A  20031014
                                          WO 2003-US32554     W  20031015
AB   The invention discloses the use of compds. and compns. of compds. that
     modulate norepinephrine levels for the treatment of vasomotor
     symptoms, e.g. thermoregulatory disorders.  The invention also discloses
     the use of compds. and compns. of compds. having norepinephrine reuptake
     inhibitor (NRI) activity alone or norepinephrine reuptake inhibitor and
     serotonin reuptake inhibitor (NRI/SRI) dual activity in combination with
     5-HT2a receptor antagonist activity.
REFERENCE COUNT:         6     THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 8 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2004:354777  CAPLUS
DOCUMENT NUMBER:         140:350602
TITLE:                   Use of norepinephrine reuptake modulators for
                         preventing and treating vasomotor symptoms
INVENTOR(S):             Deecher, Darlene Coleman; Merchenthaler, Istvan
                         Joseph; Leventhal, Liza; Sipe, Kimberly Jean;
                         O'Connor, Lawrence Thomas
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 58 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  3
PATENT INFORMATION:

     PATENT NO.          KIND    DATE          APPLICATION NO.        DATE
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     WO 2004035035        A1     20040429      WO 2003-US32760         20031015
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             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ,
             OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM,
             TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2004143008        A1     20040722      US 2003-684777          20031014
     CA 2502021           A1     20040429      CA 2003-2502021         20031015
     AU 2003282862        A1     20040504      AU 2003-282862          20031015
     EP 1551379           A1     20050713      EP 2003-774854          20031015
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     BR 2003015314        A      20050816      BR 2003-15314           20031015
     CN 1705474           A      20051207      CN 2003-80101522        20031015
     JP 2006516243        T      20060629      JP 2004-545350          20031015
PRIORITY APPLN. INFO.:                         US 2002-418591P     P  20021015
                                               US 2003-684777      A  20031014
                                               WO 2003-US32760     W  20031015
AB   The invention discloses the use of compds. and compns. of compds. that
     modulate norepinephrine levels for the prevention and treatment of
     vasomotor symptoms, e.g. hot flush, caused by, inter alia,
     thermoregulatory dysfunctions.  Compds. of the invention include e.g.
     venlafaxine.
REFERENCE COUNT:         7     THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 9 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
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ACCESSION NUMBER:    2004044305  EMBASE
TITLE:               Pediatric sleep pharmacology: You want to give my kid
                     sleeping pills?.
AUTHOR:              Pelayo R.; Chen W.; Monzon S.; Guilleminault C.
CORPORATE SOURCE:    Dr. R. Pelayo, Dept. Pediat., Psychiat., Behav. S.,
                     Stanford University, School of Medicine, 401 Quarry Road,
                     Stanford, CA 94305, United States. pelayo@stanford.edu
SOURCE:              Pediatric Clinics of North America, (2004) Vol. 51, No. 1,
                     pp. 117-134. .
                     Refs: 71
                     ISSN: 0031-3955  CODEN: PCNAA8
COUNTRY:             United States
DOCUMENT TYPE:       Journal; General Review
FILE SEGMENT:        007     Pediatrics and Pediatric Surgery
                     030     Pharmacology
                     037     Drug Literature Index
                     038     Adverse Reactions Titles
                     039     Pharmacy
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 20 Feb 2004
                     Last Updated on STN: 20 Feb 2004
AB    There is a need for greater information about the pharmacologic management
      of sleep disorders in children.  Pharmacologic guidelines must be
      developed specifically for sleep disorders in children.  Ideally, these
      guidelines should be approved by the Food and Drug Administration for a
      specific sleep disorder or for the pediatric age range.  This approval
      prevents physicians from being forced to prescribe medications as an "off
      label" indication.  Development of easy-to-swallow, chewable, or liquid
      forms of these medications would be well received by parents everywhere.
      When these are not available, instructions for compounding these
      medications into a suspension by pharmacists are needed.  Integration of
      behavioral and pharmacologic treatments may yield better patient outcomes.
      This approach requires pediatricians to have a comprehensive understanding
      of clinical sleep disorders in children.  Training programs should play
      the lead role in enhancing pediatricians' knowledge of the pharmacologic
      treatment of sleep disorders in children.

L5    ANSWER 10 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
      reserved on STN
ACCESSION NUMBER:    2003404178  EMBASE
TITLE:               [Reflex sympathetic dystrophy (complex regional pain
                     syndrome type 1), a rarely diagnosed pain syndrome in
                     childhood].
                     SYMPATHISCHE REFLEXDYSTROPHIE (COMPLEX REGIONAL PAIN
                     SYNDROME TYPE 1), EIN IM KINDESALTER SELTEN
                     DIAGNOSTIZIERTES SCHMERZVERSTARKUNGSSYNDROM.
AUTHOR:              Knirsch W.; Holder M.; Worle H.; Keimer R.; Marquard K.;
                     Stelzner J.; Kohler B.
CORPORATE SOURCE:    Dr. W. Knirsch, Padiatrisches Zentrum Olgahospital, Klin.
                     Kinderheilkunde/Jugendmedizin, Bismarckstrasse 8, 70176
                     Stuttgart, Germany. w.knirsch@olgahospital.de
SOURCE:              Monatsschrift fur Kinderheilkunde, (1 Sep 2003) Vol. 151,
                     No. 9, pp. 962-969. .
                     Refs: 35
                     ISSN: 0026-9298  CODEN: MOKIAY
COUNTRY:             Germany
DOCUMENT TYPE:       Journal; Article
FILE SEGMENT:        007     Pediatrics and Pediatric Surgery
                     019     Rehabilitation and Physical Medicine
                     032     Psychiatry
                     037     Drug Literature Index
                     038     Adverse Reactions Titles
LANGUAGE:            German
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SUMMARY LANGUAGE:    English; German
ENTRY DATE:          Entered STN: 23 Oct 2003
                     Last Updated on STN: 23 Oct 2003
AB    Case report.  We report two cases of 12 years old adolescents, who
      developed severe pain, swelling and changes in skin morphology of one
      lower limb after exertion.  Reflex sympathetic dystrophy (also called
      complex regional pain syndrome type I), a rare pain syndrome in childhood,
      was diagnosed after complete work-up.  Discussion.  Diagnosis was made by
      the trias of severe pain, autonomic vasomotor dysfunction and
      impaired motor function and confirmed by a pathologic sweat test.
      Treatment includes medicaments i.e. analgetics, non-steroid
      anti-inflammatory drugs, anti-depressants or others, physiotherapy and
      blocking of sympathetic pathways.


=> d 11-20 L5 ibib abs

L5    ANSWER 11 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER:          1999:606177  CAPLUS
DOCUMENT NUMBER:           131:295499
TITLE:                     Effect of desipramine on spontaneous
                           arterial pressure oscillations in the rat
AUTHOR(S):                 Bertram, Delphine; Barres, Christian; Julien, Claude
CORPORATE SOURCE:          Departement de Physiologie et Pharmacologie Clinique,
                           CNRS ESA 5014, Faculte de Pharmacie, Lyon, 69373, Fr.
SOURCE:                    European Journal of Pharmacology (1999), 378(3),
                           265-271
                           CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER:                 Elsevier Science B.V.
DOCUMENT TYPE:             Journal
LANGUAGE:                  English
AB    The effects of the noradrenaline uptake inhibitor desipramine (2
      mg/kg i.v., followed by 1 mg/kg every hour) on short-term variability of
      arterial blood pressure were studied in rats.  The mean arterial pressure
      was recorded in 9 conscious resting male Sprague-Dawley rats during 4
      consecutive 30-min periods: under baseline conditions, after
      desipramine administration, after cardiac autonomic blockade with
      methylatropine and atenolol, and after α-adrenoceptor blockade with
      phentolamine.  Fast Fourier transform anal. was applied to beat-to-beat
      data after resampling of consecutive 205-s time series at 10 Hz.
      Desipramine did not change the mean level and overall variability
      of the mean arterial blood pressure.  The spectral power in the
      mid-frequency (0.3-0.5 Hz) band containing the Mayer waves was decreased by
      >80% and power in the low-frequency (0.05-0.2 Hz) band was enhanced by
      .apprx.50%, especially due to the appearance of a major oscillation centered at
      0.095±0.005 Hz.  This slow oscillation was further enhanced after
      cardiac autonomic blockade and was abolished after the α-adrenergic
      blockade.  Thus, desipramine profoundly alters the short-term
      arterial pressure variability in resting rats, mainly by shifting
      vasomotor waves from 0.4 to 0.1 Hz.  Desipramine may be
      a valuable pharmacol. tool to study the dynamic aspects of arterial
      pressure control.
REFERENCE COUNT:           26    THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS
                                 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5    ANSWER 12 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
      reserved on STN
ACCESSION NUMBER:    92112857  EMBASE
DOCUMENT NUMBER:     1992112857
TITLE:               Regional variation in acute vascular homologous
                     tachyphylaxis.
AUTHOR:              Miao F.J.-P.; Lee T.J.-F.
CORPORATE SOURCE:    Department of Pharmacology, School of Medicine, Southern
                     Illinois University, P. O. Box 19230,Springfield, IL
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                         62794-9230, United States
SOURCE:                  Journal of Pharmacology and Experimental Therapeutics,
                         (1992) Vol. 260, No. 2, pp. 644-651. .
                         ISSN: 0022-3565  CODEN: JPETAB
COUNTRY:                 United States
DOCUMENT TYPE:           Journal; Article
FILE SEGMENT:            001     Anatomy, Anthropology, Embryology and Histology
                         002     Physiology
                         029     Clinical Biochemistry
                         037     Drug Literature Index
                         030     Pharmacology
LANGUAGE:                English
SUMMARY LANGUAGE:        English
ENTRY DATE:              Entered STN: 15 May 1992
                         Last Updated on STN: 15 May 1992
AB    The development of tachyphylaxis or desensitization in a tissue upon
      repeated application of an agonist is a well-established phenomenon.  To
      investigate the possible basis of vascular homologous tachyphylaxis,
      vasomotor responses in isolated cerebral and peripheral blood
      vessels from the cat, dog and rabbit upon repeated application of several
      agonists were examined using in vitro tissue bath techniques.
      Tachyphylaxis to vasodilator responses developed to repeated application
      of vasoactive intestinal polypeptide (VIP) but not to beta adrenergic
      agonist, forskolin, 8-bromo- cyclic AMP, sodium nitroprusside or
      8-bromo-cyclic GMP in feline cerebral arteries.  The tachyphylaxis to
      VIP-induced responses varied among regions and was greatest in the
      internal carotid artery (ICA), followed by the middle cerebral artery and
      least in the basilar artery (BA).  Cerebral arteries also developed
      significant tachyphylaxis to constriction induced by repeated applications
      of neuropeptide Y (NPY), alpha-1 adrenergic agonist, but not to alpha-2
      adrenergic agonist, acetylcholine (ACh) or KCl.  The tachyphylaxis to
      constrictions induced by repeated application of neuropeptide Y (NPY) also
      varied among regions, but was different from that induced by VIP; it was
      greatest in the BA, followed by the middle cerebral artery and least in
      the ICA.  Similar results were obtained in arteries without endothelial
      cells.  The density of regional innervation of NPY-immunoreactive fibers,
      which is densest in ICA and sparsest in BA, parallels that of
      VIP-immunoreactive fibers in these three regions.  There is no positive
      correlation between density of catecholamine fluorescence fibers and
      degree of tachyphylaxis to noradrenaline-induced constriction in cat ICA,
      dog saphenous arteries and rabbit ear arteries either.  These results
      indicate that development of the acute vascular homologous tachyphylaxis
      is regional and agonist specific, and is most likely related to
      characteristics of receptors rather than factors such as the density of
      innervation, endothelial function or the second messenger systems.  The
      regional variations in the development of the homologous tachyphylaxis,
      for example, to vasodilator substances, but not to constrictor substances
      in the ICA, may offer an explanation for the high incidence of
      postsubarachnoid hemorrhagic vasospasm in this region.

L5   ANSWER 13 OF 41     MEDLINE on STN
ACCESSION NUMBER:        91317538     MEDLINE
DOCUMENT NUMBER:         PubMed ID: 1860712
TITLE:                   Does regional norepinephrine spillover represent local
                         sympathetic activity?.
AUTHOR:                  Chang P C; Kriek E; van der Krogt J A; van Brummelen P
CORPORATE SOURCE:        Department of Nephrology, University of Leiden, The
                         Netherlands.
SOURCE:                  Hypertension, (1991 Jul) Vol. 18, No. 1, pp. 56-66.
                         Journal code: 7906255. ISSN: 0194-911X.
PUB. COUNTRY:            United States
DOCUMENT TYPE:           Journal; Article; (JOURNAL ARTICLE)
                         (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE:                English
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FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199109
ENTRY DATE:          Entered STN: 22 Sep 1991
                     Last Updated on STN: 22 Sep 1991
                     Entered Medline: 3 Sep 1991
AB    Regional spillover of norepinephrine (NE), based on isotope dilution and
      single-compartment steady-state kinetics, is considered one of the best
      parameters for estimating organ sympathetic activity.  However, the
      effects of local changes in clearance of NE on the spillover have not yet
      been investigated.  We studied local NE kinetics and clearance in the
      forearm of 10 healthy subjects using intra-arterial infusions of NE,
      tritiated NE, the neuronal uptake inhibitor desipramine, and
      tyramine, which competes with NE for the neuronal uptake carrier.  Before
      and during complete blockade of neuronal uptake by desipramine
      the venous concentration-time curves for tritiated NE and for NE released
      by tyramine were biexponential, consistent with the presence of (at least)
      two compartments for circulating tritiated NE and for locally released NE.
      The time constants for tyramine-induced release of NE and, in the same
      subjects during desipramine infusion, for tritiated NE were
      almost equal at the same level of forearm blood flow.  This argues against
      possible diffusion or transport differences for NE to and from the
      circulation and the synapse.  The regional intrinsic clearance capacity (a
      measure of the maximal ability of an organ to irreversibly remove drug by
      all pathways in the absence of any flow limitations) for NE decreased in
      the forearm by 65% (p less than 0.01) during neuronal uptake blockade by
      desipramine; the forearm clearance decreased by 59% (p less than
      0.001), whereas the spillover rate of NE increased from 33 +/- 5 to 63 +/-
      11 pmol.min-1 (p less than 0.05).  Nitroprusside-induced increments in
      blood flow increased the spillover of NE from 18 +/- 4 to 35 +/- 6
      pmol.min-1 (p less than 0.01); the clearance of circulating NE also
      increased (by 58%, p less than 0.05), and the intrinsic clearance capacity
      remained unchanged.  This demonstrates that regional spillover of NE is
      markedly influenced by local changes in clearance and flow.  The new
      parameter plasma appearance rate of NE is proposed.  Although also derived
      from isotope dilution, this parameter may better approximate the regional
      entry of NE into the blood pool than spillover.  This is corroborated by
      the nonsignificant changes of plasma appearance rate of NE during our
      desipramine and nitroprusside infusions.

L5    ANSWER 14 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
      reserved on STN
ACCESSION NUMBER:    90019030  EMBASE
DOCUMENT NUMBER:     1990019030
TITLE:               Differences in the central hypotensive actions of
                     α-methyldopa and clonidine in the spontaneously
                     hypertensive rat: Contribution of neurons arising from the
                     B3 and the C1 areas of the rostral ventrolateral medulla.
AUTHOR:              Drolet G.; Aslanian V.; Minson J.; Morris M.; Chalmers J.
CORPORATE SOURCE:    Department of Medicine, The Flinders University of South
                     Australia, Adelaide, SA 5042, Australia
SOURCE:              Journal of Cardiovascular Pharmacology, (1990) Vol. 15, No.
                     1, pp. 118-123. .
                     ISSN: 0160-2446  CODEN: JCPCDT
COUNTRY:             United States
DOCUMENT TYPE:       Journal; Article
FILE SEGMENT:        002     Physiology
                     018     Cardiovascular Diseases and Cardiovascular Surgery
                     029     Clinical Biochemistry
                     030     Pharmacology
                     037     Drug Literature Index
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 13 Dec 1991
                     Last Updated on STN: 13 Dec 1991
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AB The rostral ventrolateral medulla (RVLM) is the proposed site of origin of bulbospinal excitatory vasomotor neurons, and this brainstem area gives rise to chemically distinct populations of neurons, including serotonin-containing neurons of the B3 groups and epinephrine-containing neurons of the C1 group, which independently serve sympathoexcitatory functions. In the present study, we sought to establish (a) whether distinct and chemically specific pathways originating in the C1 or B3 regions are involved in the antihypertensive effects of α-methyldopa (methyldopa) and clonidine and (b) if so, whether these effects are related to an activation of α-adrenoceptors in these areas. Microinjections of methyldopa (6 nmol) or clonidine (5 nmol) were made in the C1 or B3 area in intact spontaneously hypertensive rats (SHR), pretreated with 5,7-dihydroxytryptamine (5,7-DHT) or with phentolamine. The microinjection of clonidine into both the B3 and the C1 area caused a rapid decrease in arterial pressure, whereas microinjection of methyldopa lowered the arterial pressure only after injection into the B3 area. Pretreatment with intracerebroventricular (i.c.v.) 5,7-DHT attenuated the hypotension produced by microinjection of clonidine into the B3 area, suggesting that this effect is mediated by serotonin-containing neurons. Central pretreatment with phentolamine reduced the hypotensive effects produced by injection of clonidine into either area and of methyldopa into the B3 region, consistent with previous suggestions that these central effects are mediated through α-adrenoceptors. These results suggest that both serotonin-containing and epinephrine-containing neurons contribute to the central action of clonidine, whereas the effects of methyldopa injection in RVLM appear to be mediated by serotonin-containing but not by epinephrine-containing neurons.

L5 ANSWER 15 OF 41 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 2

ACCESSION NUMBER: 88158502 EMBASE
DOCUMENT NUMBER: 1988158502
TITLE: Prepontine knife cut-induced hyperthermia in the rat. Effect of chemical sympathectomy and surgical denervation of brown adipose tissue.
AUTHOR: Benzi R.H.; Shibata M.; Seydoux J.; Girardier L.
CORPORATE SOURCE: Department of Physiology, University Medical Center, CH-1211 Geneva 4, Switzerland
SOURCE: Pflugers Archiv European Journal of Physiology, (1988) Vol. 411, No. 6, pp. 593-599. .
ISSN: 0031-6768 CODEN: PFLABK
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 002 Physiology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 11 Dec 1991
Last Updated on STN: 11 Dec 1991

AB The effect of brown adipose tissue (BAT) denervation on the prepontine knife cut-induced hyperthermia was studied. The knife cut has earlier been shown to induce a steady state hyperthermia of 3 to 4°C, as a result of marked activation of the BAT. Before the lesion, the interscapular BAT (IBAT) temperature was lower than the colonic temperature, but the temperature gradient reversed a few minutes after the lesion and the fractional blood flow increased 12-fold. Bilateral sectioning of the 5 nerves supplying IBAT did not modify either the magnitude or the kinetics of the IBAT hyperthermic response. The IBAT fractional blood flow, which was 15 times higher in denervated than in intact tissue before the lesion, scarcely increased following the lesion despite the sharp increase in the tissue's metabolic activity. Chemical sympathectomy with 6-hydroxydopamine suppressed the hyperthermic response. Propranolol or hexamethonium injected i.v. during the steady state hyperthermia resulted in rapid drop in IBAT temperature and in a reversal of the gradient between IBAT and colonic temperature both in denervated and in intact IBAT. Injection of desipramine, an inhibitor of noradrenaline reuptake, resulted in itself in an increase of temperature in both intact and denervated tissue, which is circumstantial evidence for the presence of a functional residual innervation in the latter. These results indicate that vasomotor fibres run within intercostal nerves supplying IBAT and since severing of these nerves did not prevent the sympathetic stimulation of the tissue, it can be inferred that the residual innervation entering the IBAT along with the blood vessels is conveying stimulating signals originating in peripheral structures. The observations made by Moragas and Toran (1985) of a very active lipolysis in BAT from anencephalic infants are discussed in the context of these findings.

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ACCESSION NUMBER: 87085434 EMBASE
DOCUMENT NUMBER: 1987085434
TITLE: Bombesin-induced hypothermia: Possible involvement of cholinergic and dopaminergic receptors in the rat hypothalamus.
AUTHOR: Chiu W.-T.; Lin L.-S.; Shih C.-J.; Lin M.-T.
CORPORATE SOURCE: Division of Neurosurgery, Department of Surgery, MacKay Memorial Hospital, Taipei, Taiwan,Province of China
SOURCE: Experimental Neurology, (1987) Vol. 95, No. 2, pp. 368-377.
.
CODEN: EXNEAC
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT:

037	Drug Literature Index
002	Physiology
008	Neurology and Neurosurgery
030	Pharmacology

LANGUAGE: English
ENTRY DATE: Entered STN: 11 Dec 1991
Last Updated on STN: 11 Dec 1991

AB The thermal responses of rats which were pretreated with 5,7-dihydroxytryptamine to deplete hypothalamic 5-hydroxytryptamine, 6-hydroxydopamine to deplete hypothalamic catecholamines, phentolamine and propranolol to inhibit adrenergic receptors, haloperidol to inhibit dopamine receptors, or atropine to inhibit cholinergic receptors, to intrahypothalamic administration of bombesin were compared with those of control rats. The bombesin-induced hypothermia was attenuated by pretreatment of the rats with either hypothalamic dopamine depletion or receptor blockade, or hypothalamic cholinergic receptor blockade. The reduction in the bombesin-induced hypothermia in the treated rats was due to the reduction of metabolic and vasomotor response. The data indicate that bombesin may act on hypothalamic dopamine and/or cholinergic receptor mechanisms to induce hypothermia by promoting a reduction in metabolic heat production and an enhancement in heat loss in rats.

L5 ANSWER 17 OF 41 MEDLINE on STN

ACCESSION NUMBER: 86001699 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3930004
TITLE: Regional depletion of central nervous system catecholamines: effects on blood pressure and drinking behavior.
AUTHOR: Gordon F J; Brody M J; Johnson A K
CONTRACT NUMBER: GN-07069 (NHLBI)
HL-07121 (NHLBI)
HLP-14388
+
SOURCE: Brain research, (1985 Oct 21) Vol. 345, No. 2, pp. 285-97.
Journal code: 0045503. ISSN: 0006-8993.
PUB. COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198511
ENTRY DATE: Entered STN: 21 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 20 Nov 1985

AB The purpose of the present study was to identify which catecholamine-containing neurons (norepinephrine (NE) or dopamine (DA)) and which central nervous system (CNS) region(s) innervated by them might participate in the pressor and drinking responses produced by central drug stimulation. Forebrain NE was reduced in rats by injecting 4 micrograms of 6-hydroxydopamine (6-OHDA) into the ascending noradrenergic bundles. Spinal cord NE was depleted by intracisternal injection of 50 micrograms 6-OHDA. Depletion of forebrain DA was produced by bilateral injection of 4 micrograms 6-OHDA into the substantia nigra of desipramine -pretreated rats. Pressor responses to various doses of angiotensin II (AII), carbachol or hyperosmolar NaCl injected into the lateral ventricles (LVT); and drinking responses to LVT AII and carbachol were examined. Injection of 6-OHDA into the noradrenergic bundles reduced telencephalic and hypothalamic NE by more than 80% without significantly affecting brain DA or spinal cord NE. Intracisternal 6-OHDA depleted spinal cord NE by 80% and forebrain NE by 20-25% without reducing brain DA. Injection of 6-OHDA into the substantia nigra reduced telencephalic DA by 86% and NE by 29% without significantly affecting NE in other CNS regions. Substantia nigra 6-OHDA injected animals evidenced attenuated drinking to both LVT AII and carbachol. Pressor responses to LVT AII, carbachol and hypertonic saline were largely unaffected. Almost complete depletion of brain and/or spinal cord NE failed to alter centrally mediated drinking or pressor responses. These data indicate that the integrity of brain DA neurons is required for the behavioral but not hypertensive responses produced by central drug stimulation.

L5 ANSWER 18 OF 41 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 85168297 EMBASE
DOCUMENT NUMBER: 1985168297
TITLE: Cholecystokinin-induced hypothermia: Possible involvement of serotoninergic mechanisms in the rat hypothalamus.
AUTHOR: Liu H.J.; Lin M.T.
CORPORATE SOURCE: Department of Physiology, Taipei Medical College, Taipei, Taiwan,Province of China
SOURCE: Pharmacology, (1985) Vol. 31, No. 2, pp. 108-114. .
CODEN: PHMGBN
COUNTRY: Switzerland
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
003 Endocrinology
030 Pharmacology
002 Physiology
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB The thermal effects of rats which were pretreated with 5,7-dihydroxytryptamine to deplete hypothalamic 5-hydroxytryptamine (5-HT) or with a 5-HT receptor blocker to intrahypothalamic administration of cholecystokinin (CCK) were compared with those of control rats. The CCK-induced hypothermic response was attenuated by pretreatment of the rats with either hypothalamic 5-HT depletion or receptor blockade. The reduction in the CCK hypothermia in the treated rats was due to the reduction of metabolic and vasomotor response. The data indicate that CCK may act on a 5-HT pathway within the hypothalamus to induce its hypothermia by promoting a reduction in metabolic heat

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     production and an enhancement in heat loss in rats.

L5   ANSWER 19 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    84059458  EMBASE
DOCUMENT NUMBER:     1984059458
TITLE:               Adrenaline mediates a positive feedback loop in
                     noradrenergic transmission: Its possible role in
                     development of hypertension.
AUTHOR:              Rand M.J.; Majewski H.
CORPORATE SOURCE:    Department of Pharmacology, University of Melbourne,
                     Melbourne, Vic. 3052, Australia
SOURCE:              Clinical and Experimental Hypertension - Part A Theory and
                     Practice, (1984) Vol. 6, No. 1-2, pp. 347-370. .
                     CODEN: CEHADM
COUNTRY:             United States
DOCUMENT TYPE:       Journal
FILE SEGMENT:        037     Drug Literature Index
                     030     Pharmacology
                     018     Cardiovascular Diseases and Cardiovascular Surgery
                     008     Neurology and Neurosurgery
                     003     Endocrinology
LANGUAGE:            English
ENTRY DATE:          Entered STN: 10 Dec 1991
                     Last Updated on STN: 10 Dec 1991
AB   Adrenaline activates prejunctional β-adrenoceptors of the
     β2-subtype on sympathetic nerve terminals and enhances noradrenergic
     transmission.  Adrenaline can be incorporated in transmitter stores of
     noradrenergic nerves and, when released as a cotransmitter, activates the
     prejunctional β2-adrenoceptors, thereby mediating autofacilitation of
     noradrenergic transmission.  Adrenaline released from the adrenal medulla
     in stress may be incorporated in noradrenergic transmitter stores and
     reach a sufficient concentration as a cotransmitter to activate the
     autofacilitatory feedback loop involving prejunctional
     β2-adrenoceptors, resulting in prolongation of the increases in
     vasomotor tone and cardiac activity that occur acutely: with
     frequent repetition of stress, there may be progression into a
     hypertensive state.  In accord with this hypothesis, adrenaline
     administration produces persistent increases in blood pressure in rats,
     and plasma levels of adrenaline are elevated in a proportion of
     hypertensive patients; furthermore, repeated stress produces prolonged
     increases in blood pressure in animals and man.

L5   ANSWER 20 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    84166297  EMBASE
DOCUMENT NUMBER:     1984166297
TITLE:               Clonidine-induced hypothermia: Possible involvement of
                     cholinergic and serotonergic mechanisms.
AUTHOR:              Lin M.T.; Shian L.R.; Leu S.Y.
CORPORATE SOURCE:    Department of Physiology and Biophysics, National Defense
                     Medical Center, Taipei, Taiwan,Province of China
SOURCE:              Naunyn-Schmiedeberg's Archives of Pharmacology, (1984) Vol.
                     326, No. 2, pp. 124-128. .
                     CODEN: NSAPCC
COUNTRY:             Germany
DOCUMENT TYPE:       Journal
FILE SEGMENT:        037     Drug Literature Index
                     030     Pharmacology
LANGUAGE:            English
ENTRY DATE:          Entered STN: 10 Dec 1991
                     Last Updated on STN: 10 Dec 1991
AB   1.  The thermoregulatory effects (including metabolic, vasomotor
     and repiratory activities) produced by an injection of clonidine (1 - 3
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          µg in 0.5 µl) into the preoptic anterior hypothalamus were assessed
          in conscious rats at ambient temperatures (T(a)) of 8, 22, and
          30°C. 2.  Intrahypothalamic administration of clonidine caused a
          dose-dependent fall in rectal temperature at T(a) 8°C and
          22°C.  The hypothermia in response to clonidine was due to
          decreased metabolic heat production and/or cutaneous vasodilation.  There
          was no change in respiratory evaporative heat loss. 3.  The
          clonidine-induced hypothermic response was attenuated by pretreatment of
          the rats with either 5,7-dihydroxytryptamine (10 µg, administered
          intrahypothalamicly, 14 days before clonidine injection), yohimbine (0.2
          µg, administered intrahypothalamicly, 10 min before clonidine
          injection), cyproheptadine (1  g, administered intrahypothalamicly, 10 min
          before clonidine injection), or atropine (0.1 µg, administered
          intrahypothalamicly, 10 min before clonidine injection). 4.  The data
          indicate that clonidine may act on α-adrenoceptors located on a
          serotonin-acetylcholine pathway within the preoptic anterior hypothalamus
          to induce hypothermia by promoting a reduction in metabolic heat
          production and/or an enhancement in dry heat loss in rats.


=> s L5 and 21-30 ibib abs
L11           0 L5 AND 21-30 IBIB ABS

=>  d L5 and 21-30 ibib abs
'AND' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):d L5 21-30 ibib abs
'D' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> d L5 21-30 ibib abs

L5   ANSWER 21 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN DUPLICATE 3
ACCESSION NUMBER:         1983:464990  CAPLUS
DOCUMENT NUMBER:          99:64990
TITLE:                    Evidence for a bulbospinal serotonergic pressor
                          pathway in the rat brain
AUTHOR(S):                Howe, P. R. C.; Kuhn, D. M.; Minson, J. B.; Stead, B.
                          H.; Chalmers, J. P.
CORPORATE SOURCE:         Cent. Neurosci., Flinders Univ. South Australia,
                          Bedford Park, Australia
SOURCE:                   Brain Research (1983), 270(1), 29-36
                          CODEN: BRREAP; ISSN: 0006-8993
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   The cardiovascular role of spinal serotoninergic neurons descending from
     cells near the ventrolateral surface of the medulla oblongata was
     investigated by stimulating these cells in normal animals and in animals
     with selective chemical ablation of serotoninergic nerves.
     5,7-Dihydroxytryptamine (5,7DHT) was injected into the lateral cerebral
     ventricle (i.c.v.) to produce a generalized destruction of central
     serotoninergic pathways, with preliminary i.p. administration of
     desipramine to prevent depletion of noradrenaline stores.  In
     other expts., 5,7-DHT was injected directly into the cervical spinal cord,
     after preliminary treatment with desipramine, to produce
     selective destruction of spinal serotoninergic nerves which was confirmed
     both biochem. and immunohistochem.  Elec. stimulation near the lateral
     serotoninergic cells in the B1 and B3 cell groups of the medulla elicited
     pressor responses in control rats.  Microinjections of kainic acid
     [487-79-6] or L-glutamate  [56-86-0] at the same sites also produced an
     increase in mean arterial pressure.  Selective destruction of
     serotoninergic nerves, whether produced by i.c.v. or intraspinal
     administration of 5,7-DHT, reduced the magnitude of the pressor response
     to elec. stimulation by >50%.  Thus, serotoninergic nerve cells adjacent
     to the ventrolateral surface of the medulla oblongata and projecting to
     the intermediolateral cell column serve to elevate arterial pressure and
     maintain vasomotor tone.

L5   ANSWER 22 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    83093071  EMBASE
DOCUMENT NUMBER:     1983093071
TITLE:               Adrenomedullary secretory response to midbrain stimulation
                     in rat: Effects of depletion of brain catecholamines or
                     serotinin.
AUTHOR:              Gauthier P.; Reader T.A.
CORPORATE SOURCE:    Dep. Physiol., Cent. Rech. Sci. Neurol., Fac. Med., Univ.
                     Montreal, Montreal, Que. H3C 3T8, Canada
SOURCE:              Canadian Journal of Physiology and Pharmacology, (1982)
                     Vol. 60, No. 12, pp. 1464-1474. .
                     CODEN: CJPPA3
COUNTRY:             Canada
DOCUMENT TYPE:       Journal
FILE SEGMENT:        037     Drug Literature Index
                     030     Pharmacology
                     008     Neurology and Neurosurgery
                     003     Endocrinology
LANGUAGE:            English
SUMMARY LANGUAGE:    French
ENTRY DATE:          Entered STN: 9 Dec 1991
                     Last Updated on STN: 9 Dec 1991
AB   Electrical stimulation of the periaqueductal gray substance (PAG) of the
     rostral midbrain of the rat produced biphasic or monophasic pressure
     responses depending on the duration of the stimulus train.  Marked
     increases in plasma noradrenaline (NA) and adrenaline (A) levels
     accompanied the pressor responses, indicating the participation of the
     adrenal medulla.  Depletion of central catecholamines (CA) by
     intraventricular administration of 6-hydroxydopamine (6-OHDA) did not
     affect the primary vasomotor component but markedly depleted
     adrenal CA levels and attenuated the adrenomedullary component of the
     response to brain stimulation.  The intraperitoneal administration of
     p-chlorophenylalanine (pCPA) not only depleted brain serotonin (5-HT)
     levels but also reduced CA levels significantly.  The adrenaline (A)
     levels were reduced in the adrenal glands of these rats and the adrenal
     secretory response to brain stimulation was attenuated.  In contrast, the
     selective destruction of central 5-HT neurons by intraventricular
     injection of 5,7-dihydroxytryptamine (5,7-DHT) in rats pretreated with
     desmethylimipramine (DMI) did not influence either the pressor nor the
     plasma CA responses to brain stimulation.  Furthermore, the adrenal glands
     of these rats were normal.  The results suggest that: (1) the central
     catecholaminergic neurons play an important role in the regulation of the
     adrenal glands but are not essential for the activation of the sympathetic
     vasconstrictor fiber system; (2) the pressor and plasma CA responses to
     PAG stimulation are not dependent on the central serotonergic system.

L5   ANSWER 23 OF 41     MEDLINE on STN
ACCESSION NUMBER:    82232705     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 7093584
TITLE:               Benztropine-induced prolongation of responses to
                     vasodilator nerve stimulation in the canine paw pad.
AUTHOR:              Bell C
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SOURCE: British journal of pharmacology, (1982 Jun) Vol. 76, No. 2, pp. 231-3.
Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198209
ENTRY DATE: Entered STN: 17 Mar 1990
Last Updated on STN: 17 Mar 1990
Entered Medline: 24 Sep 1982

AB After blockade of noradrenergic transmission with guanethidine in anaesthetized dogs, stimulation of the peripheral end of the cut tibial nerve (1 Hz, 10 pulses) produces a vasodilator response which is localized to the circulation of the paw pads. The time course of the response is considerably prolonged after systemic administration of the dopamine-uptake blocking drug, benztropine (1 mg/kg). This effect is not due to inhibition of Uptake 1, as benztropine, unlike desmethylimipramine, does not prolong responses to noradrenergic vasoconstrictor nerve stimulation. The results support previous evidence suggesting that the dilator response to tibial nerve stimulation involves neural release of dopamine.

L5 ANSWER 24 OF 41 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 82134513 EMBASE
DOCUMENT NUMBER: 1982134513
TITLE: Studies on β-adrenoceptor mediated facilitation of sympathetic neurotransmission.
AUTHOR: Dahlof C.
CORPORATE SOURCE: Res. Lab., Dept. Pharmacol., AB Hassle, S-431 83 Molndal, Sweden
SOURCE: Acta Physiologica Scandinavica, (1981) Vol. 113, No. Suppl. 500, pp. 147p. .
CODEN: APSCAX
COUNTRY: Sweden
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
002 Physiology
008 Neurology and Neurosurgery
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB The series of studies summarized in this thesis was initiated by the finding that the β-adrenoceptor antagonist propranolol potentiated the vasoconstrictor response to exogenous noradrenaline(NA), but attenuated the response to sympathetic vasomotor nerve stimulation in the perfused hind limb of the anaesthetized cat. The nerve terminals of the peripheral sympathetic nerve system appear to be endowed with β-adrenoceptors of both the β1- and β2-subtype.
Activation of these presynaptic β-adrenoceptors by exogenously or endogenously released agonists leads to an increase of the amount of noradrenaline(NA) released per nerve impulse. The action of β-adrenoceptor agonists appears to be stereospecific and the mediated effect on transmitter release is most pronounced at low frequency nerve stimulation. Facilitation of sympathetic nerve stimulation (SNS) induced transmitter release in the presence of β-adrenoceptor agonists has been found in many, but not all, tissues studied. The mechanism by which β-adrenoceptors increase transmitter release is unknown. One possible way that β-adrenoceptor agonists facilitate the stimulation induced release of NA could be via activation of neuronal adenylate cyclase leading to formation of intraneuronal cAMP. It seems possible

that presynaptic facilitatory β-adrenoceptors can be activated by low levels of circulating adrenaline(A). Apparently the adrenal medullary A can also be taken up from the circulation into the adrenergic nerve endings and released as co-transmitter during nervous activity. Thus both A and NA seem to be involved in a physiological control mechanism mediated by facilitatory β1- and β2-adrenoceptors. Inhibition of presynaptic β-adrenoceptors causes an acute depression of the release of NA per impulse resulting in an unchanged or slightly reduced effector response. The activity of noradrenergic neurones in the brain of the rat seems to be subject to a stimulatory β2-adrenergic control mechanism. The effect of inhibition of these central β2-adrenoceptors appears to be delayed. This might indicate a trophic nature of this control mechanism.

L5 ANSWER 25 OF 41 MEDLINE on STN

ACCESSION NUMBER:	81059765 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 6254375
TITLE:	Evaluation of presynaptic alpha-receptor function in the canine renal vascular bed.
AUTHOR:	Robie N W
CONTRACT NUMBER:	5-S07-RR-5376-16 (NCRR)
SOURCE:	The American journal of physiology, (1980 Sep) Vol. 239, No. 3, pp. H422-6. Journal code: 0370511. ISSN: 0002-9513.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	198101
ENTRY DATE:	Entered STN: 16 Mar 1990 Last Updated on STN: 3 Feb 1997 Entered Medline: 26 Jan 1981

AB The functional significance of presynaptic alpha-receptor modulation of sympathetic nerves was examined in vivo in the canine renal vascular bed. In pentobarbital-anesthetized dogs, the vasoconstrictor response to renal nerve stimulation and exogenous norepinephrine was compared before and during intra-arterial infusions of epinephrine, oxymetazoline, clonidine, and norepinephrine. Only epinephrine produced a modest decrease in stimulation-induced vasoconstriction at 1 Hz. After pretreatment with desipramine, intra-arterial infusions of epinephrine or norepinephrine did not alter stimulation-induced vasoconstrictor responses relative to exogenous norepinephrine. Further, neither yohimbine nor phentolamine (10(-9) to 10(-3) g, intra-arterial) produced a distinctly increased vasoconstrictor response to nerve stimulation relative to exogenous norepinephrine. Thus, studies using alpha-receptor agonists, antagonists, and inhibition of neuronal uptake failed to reveal a physiologically significant alpha-receptor-mediated negative feedback mechanism for stimulation-induced vasoconstriction in the canine renal vascular bed.

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ACCESSION NUMBER:	80228113 EMBASE
DOCUMENT NUMBER:	1980228113
TITLE:	Effects of methacholine and acetylcholine on metabolic, respiratory, vasomotor and body temperature responses in rabbits.
AUTHOR:	Chen F.F.; Lin M.T.
CORPORATE SOURCE:	Dept. Physiol. Biophys., Nat. Def. Med. Cent., Taipei, Italy
SOURCE:	Pharmacology, (1980) Vol. 21, No. 5, pp. 333-341. . CODEN: PHMGBN

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COUNTRY:               Switzerland
DOCUMENT TYPE:         Journal
FILE SEGMENT:          037     Drug Literature Index
                       030     Pharmacology
                       002     Physiology
LANGUAGE:              English
ENTRY DATE:            Entered STN: 9 Dec 1991
                       Last Updated on STN: 9 Dec 1991
AB    Central administration methacholine and acetylcholine each produced a
      dose-dependent hypothermia in conscious rabbits at room temperature
      (22°C) and blow.  At room temperature, hypothermia in response to
      methacholine and acetylcholine was due to decreased metabolism and
      increased heat loss (both an increase in ear skin blood flow and in
      respiratory evaporative heat loss).  In the cold (4°C), hypothermia
      was brought about by a decrease in metabolic heat production and an
      increase in respiratory evaporative heat loss.  However, in the heat
      (32°C), central administration of methacholine and acetylcholine
      each produced a dose-dependent hyperthermia in rabbits.  The hyperthermia
      was due to a decrease in both ear skin blood flow and respiratory
      evaporative heat loss.  Furthermore, the effects of methacholine of
      acetylcholine on body temperature was antagonized by pretreatment with
      atropine.

L5    ANSWER 27 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
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ACCESSION NUMBER:      81044688  EMBASE
DOCUMENT NUMBER:       1981044688
TITLE:                 Tail temperature in rats after intraventricular injections
                       of 6-hydroxydopamine or 5,7-dihydroxy-tryptamine.
AUTHOR:                Ishikawa Y.; Matuszek M.; Nakayama T.
CORPORATE SOURCE:      Dept. Physiol., Osaka Univ. Med. Sch., Osaka 530, Japan
SOURCE:                Neuroscience Letters, (1980) Vol. 20, No. 2, pp. 177-181. .
                       CODEN: NELED5
COUNTRY:               Ireland
DOCUMENT TYPE:         Journal
FILE SEGMENT:          002     Physiology
                       037     Drug Literature Index
LANGUAGE:              English
ENTRY DATE:            Entered STN: 9 Dec 1991
                       Last Updated on STN: 9 Dec 1991
AB    The tail vasomotor tone in rats is reduced by the
      intraventricular injection of 5,7-dihydroxytryptamine but not by
      6-hydroxydopamine.  Central serotonergic but not noradrenergic neurons
      participate in vasoconstriction of the tail.

L5    ANSWER 28 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
      reserved on STN
ACCESSION NUMBER:      79048278  EMBASE
DOCUMENT NUMBER:       1979048278
TITLE:                 Involvement of uptake1 and uptake2 in terminating the
                       cardiovascular activity of noradrenaline in normotensive
                       and genetically hypertensive rats.
AUTHOR:                Bell C.; Kushinsky R.
CORPORATE SOURCE:      Dept. Physiol., Univ. Melbourne, Parkville, Victoria,
                       Australia
SOURCE:                Journal of Physiology, (1978) Vol. Vol. 283, pp. 41-51. .
                       CODEN: JPHYA7
COUNTRY:               United Kingdom
DOCUMENT TYPE:         Journal
FILE SEGMENT:          037     Drug Literature Index
                       002     Physiology
LANGUAGE:              English
AB    In pithed, pancuronium-treated rats, inhibition of Uptake 1 with
      desmethylimipramine (2.5 mg/kg i.v.) increased the time course of the
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     pressor response to vasomotor nerve stimulation (1 and 5 Hz for
     5 sec) but not the time course of the pressor response to injected
     noradrenaline (10 and 50 ng i.v.).  Subsequent inhibition of Uptake 2 with
     metanephrine (3 mg/kg, i.v.) did not affect the time course of responses
     to either vasomotor nerve stimulation or injected noradrenaline.
     It is concluded that Uptake 1 but not Uptake 2 is important in terminating
     the activity of noradrenaline released from rat vasomotor nerves
     and that neither process inactivates intraluminal catecholamine.  By
     contrast, chronotropic responses of rat atria to adrenergic nerve
     stimulation were prolonged by blockade of both Uptake 1 and Uptake 2, in
     agreement with previous evidence for involvement of both processess in
     terminating sino-atrial adrenergic responses.  Animals from the Otago
     strain of genetically hypertensive animals were identical to normotensives
     in their vascular handling of catecholamine.  However, they lacked an
     atrial Uptake 2 process.  This defect may be related to the high resting
     heart rate and abnormal cardiac catecholamine turnover observed to exist
     in the Otago hypertensive rats.

L5   ANSWER 29 OF 41     MEDLINE on STN
ACCESSION NUMBER:    78081436     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 202357
TITLE:               Relaxation of smooth muscle following contraction elicited
                     by sympathetic nerve stimulation in vivo.
AUTHOR:              Eccles R; MacLean A G
SOURCE:              British journal of pharmacology, (1977 Dec) Vol. 61, No. 4,
                     pp. 551-8.
                     Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY:        ENGLAND: United Kingdom
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         197803
ENTRY DATE:          Entered STN: 14 Mar 1990
                     Last Updated on STN: 14 Mar 1990
                     Entered Medline: 10 Mar 1978
AB   1.  A method for studying in vivo the process of neuroeffector
     transmission in the nictitating membrane and nasal blood vessels of the
     cat is described. 2.  Administration of desmethylimipramine or cocaine
     caused increases in both the amplitude and duration of the nasal and
     membrane responses which may be explained by inhibition of neuronal uptake
     of noradrenaline. 3.  Phenoxybenzamine depressed the responses to nerve
     stimulation, but had little effect on the relationship between response
     amplitude and rate of recovery. 4.  The relationship between response
     amplitude and rate of recovery is discussed and related to the sigmoid
     shape of a log concentration-response curve.

L5   ANSWER 30 OF 41     MEDLINE on STN
ACCESSION NUMBER:    76240209     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 950610
TITLE:               Action of sympathetic nerves of inner and outer muscle of
                     sheep carotid artery, and effect of pressure on nerve
                     distribution.
AUTHOR:              Keatinge W R; Torrie C
SOURCE:              The Journal of physiology, (1976 Jun) Vol. 257, No. 3, pp.
                     699-712.
                     Journal code: 0266262. ISSN: 0022-3751.
PUB. COUNTRY:        ENGLAND: United Kingdom
DOCUMENT TYPE:       (IN VITRO)
                     Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         197610
ENTRY DATE:          Entered STN: 13 Mar 1990
                     Last Updated on STN: 13 Mar 1990
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                    Entered Medline: 2 Oct 1976
AB    1.  The direction of torsion produced during active shortening of helical
      strips of sheep carotid arteries was measured to assess whether inner or
      outer muscle was contracting. 2.  Noradrenaline contracted inner
      (non-innervated) muscle in lower concentrations than were needed to
      contract outer (innervated) muscle, even with desipramine
      present to prevent uptake of noradrenaline by the nerves and with enough
      cyanide present to rise the normally low O2 tension of inner muscle to
      that of outer muscle. 3.  Activation of sympathetic nerves in the outer
      part of the artery by nicotine caused almost evenly balanced contraction
      of both parts of the wall, with slight bias to outer contraction. 4.
      Moderate external constriction of the artery in vivo for 10-17 days, in
      order to raise pressure throughout the wall to intraluminal pressure, made
      the entire wall nerve-free. 5.  The results provide evidence that the
      nerves can induce substantial activation of inner muscle, which is highly
      sensitive to noradrenaline, and that the absence of nerves from inner
      muscle can be explained by the high pressure there.


=> d L5 31-41 ibib abs

L5    ANSWER 31 OF 41     MEDLINE on STN
ACCESSION NUMBER:   76208624     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 1277402
TITLE:              Neurogenic sympathetic vasoconstriction of the rabbit
                    basilar artery.
AUTHOR:             Lee T J; Su C; Bevan J A
SOURCE:             Circulation research, (1976 Jul) Vol. 39, No. 1, pp. 120-6.
                    Journal code: 0047103. ISSN: 0009-7330.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        197608
ENTRY DATE:         Entered STN: 13 Mar 1990
                    Last Updated on STN: 13 Mar 1990
                    Entered Medline: 23 Aug 1976
AB    When examined by fluorescence microscopy the rabbit basilar artery
      contains a rich adrenergic-like plexus at the adventitiomedial junction.
      The fluorescence disappears upon chronic reserpinization and bilateral
      superior cervical ganglionectomy.  Transmural stimulation of intramural
      nerves a results in a response which is predominantly constrictor but also
      contains a small, inconstant dilator component.  The constrictor response
      is abolished by chronic reserpinization, bilateral superior cervical
      ganglionectomy, and cold storage of the preparation.  The constriction is
      prevented by the adrenergic neuron blocking agents guanethidine and
      bretylium but not by such alpha-adrenergic receptor blocking agents as
      phenoxybenzamine (PBZ), phentolamine, and tolazoline.  Our results show
      that doses of the three latter agents sufficient to abolish contractions
      to norepinephrine (NE) in concentrations of up to 10(-2) M only potentiate
      and prolong the contractile response to nerve stimulation.  The
      beta-adrenergic receptor blocking agent, propranolol, and inhibitors of NE
      neuronal uptake, such as desipramine (desmethylimipramine, DMI)
      and cocaine, do not influence the size of the neurogenic response.  These
      results suggest that the vasoconstrictor component of the rabbit basilar
      artery response to transmural nerve stimulation (TNS) is mediated via
      sympathetic adrenergic-like neurons, but at the same time also raise the
      question whether the transmission process is typical of classic adrenergic
      neuroeffector mechanisms.

L5    ANSWER 32 OF 41     MEDLINE on STN                    DUPLICATE 4
ACCESSION NUMBER:   76037026     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 1180090
TITLE:              Vasomotor nerve control of isolated arteries and
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                    veins.
AUTHOR:             Ljung B; Bevan J A; Pegram B L; Purdy R E; Su M
SOURCE:             Acta physiologica Scandinavica, (1975 Aug) Vol. 94, No. 4,
                    pp. 506-16.
                    Journal code: 0370362. ISSN: 0001-6772.
PUB. COUNTRY:       Sweden
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        197512
ENTRY DATE:         Entered STN: 13 Mar 1990
                    Last Updated on STN: 13 Mar 1990
                    Entered Medline: 18 Dec 1975
AB    In order to compare neuro-effector function in different blood vessels,
      frequency-response relationships were determined for the following
      preparations: 1)Isolated rings of the proximal saphenous, distal saphenous
      and ear arteries, the parietal branch of the internal iliac vein and the
      small saphenous vein from the rabbit, 2) spiral strips of the rabbit
      pulmonary artery and 3) longitudinal preparations of the rat portal vein.
      In each rabbit tissue only one low (less than or equal to 4 Hz) and one
      high (larger than or equal to 8 Hz) transmural nerve stimulation frequency
      was applied until steady state responses were obtained and these were
      expressed as a percentage of a maximum response to exogenous noradrenaline
      (NA) applied in each experiment.  The general shape of the
      frequency-response curves was similar, but differences in steepness and
      amplitude of the maximum neurogenic response relative to exogenous NA were
      found.  The steepness of the frequency-response relationships of the veins
      tended to be greater than those of the arteries.  It appears that factors
      such as close neuro-muscular contacts, presence of terminal nerve fibres
      within the media and the operation of mechanisms for myogenic propagation
      of activity contribute to the effectiveness of neurogenic vascular control
      as revealed by frequency-response curves.  In vivo, geometrical factors
      can greatly augment the hemodynamic significance of the observed
      differences.

L5    ANSWER 33 OF 41  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
      reserved on STN
ACCESSION NUMBER:   74176826  EMBASE
DOCUMENT NUMBER:    1974176826
TITLE:              Evidence for bulbospinal control of sympathetic
                    preganglionic neurons by monoaminergic pathways.
AUTHOR:             Neumayr R.J.; Hare B.D.; Franz D.N.
CORPORATE SOURCE:   Dept. Pharmacol., Univ. Utah, Salt Lake City, Ut. 84132,
                    United States
SOURCE:             Life Sciences, (1974) Vol. 14, No. 4, pp. 793-806. .
                    CODEN: LIFSAK
DOCUMENT TYPE:      Journal
FILE SEGMENT:       037     Drug Literature Index
                    002     Physiology
                    008     Neurology and Neurosurgery
LANGUAGE:           English
AB    Medullary pressor and depressor areas in the cat showed close correlations
      with the locations of bulbospinal, NE and 5 HT perikarya, respectively.
      Microelectrode stimulation of these areas evoked or inhibited sympathetic
      discharges recorded from preganglionic rami.  Discharges were also evoked
      in spinal cats by stimulation in a restricted area of the lateral
      funiculus of the cervical spinal cord.  Pharmacologic manipulation of
      evoked discharges by monoamine precursors, chlorpromazine, and
      desipramine support the proposal that sympathetic preganglionic
      neurons are reciprocally controlled by bulbospinal NE excitatory and 5 HT
      inhibitory pathways.

L5    ANSWER 34 OF 41     MEDLINE on STN
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ACCESSION NUMBER:     74297395     MEDLINE
DOCUMENT NUMBER:      PubMed ID: 4368325
TITLE:                A differential effect of chronically administered
                      guanethidine on sympathetic neurotransmission to the dog
                      heart and mesenteric arterial blood vessels.
AUTHOR:               Clarke D E; Jandhyala B S; Cavero I; Dixit B N; Buckley J P
SOURCE:               Canadian journal of physiology and pharmacology, (1974 Jun)
                      Vol. 52, No. 3, pp. 641-8.
                      Journal code: 0372712. ISSN: 0008-4212.
PUB. COUNTRY:         Canada
DOCUMENT TYPE:        (IN VITRO)
                      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:             English
FILE SEGMENT:         Priority Journals
ENTRY MONTH:          197410
ENTRY DATE:           Entered STN: 10 Mar 1990
                      Last Updated on STN: 10 Mar 1990
                      Entered Medline: 31 Oct 1974

L5   ANSWER 35 OF 41     MEDLINE on STN
ACCESSION NUMBER:     72158000     MEDLINE
DOCUMENT NUMBER:      PubMed ID: 4336047
TITLE:                Release of endogenous noradrenaline from an isolated
                      muscular artery. Release of endogenous noradrenaline from
                      an isolated muscular artery.
AUTHOR:               Bell C; Vogt M
SOURCE:               The Journal of physiology, (1971 Jun) Vol. 215, No. 2, pp.
                      509-20.
                      Journal code: 0266262. ISSN: 0022-3751.
PUB. COUNTRY:         ENGLAND: United Kingdom
DOCUMENT TYPE:        Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:             English
FILE SEGMENT:         Priority Journals
ENTRY MONTH:          197206
ENTRY DATE:           Entered STN: 10 Mar 1990
                      Last Updated on STN: 10 Mar 1990
                      Entered Medline: 19 Jun 1972

L5   ANSWER 36 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:            1971:51884  CAPLUS
DOCUMENT NUMBER:             74:51884
TITLE:                       Potentiating action of desmethylimipramine on the
                             cardiovascular effects of noradrenaline and some other
                             pressor agents
AUTHOR(S):                   Astrom, A.
CORPORATE SOURCE:            Dep. Physiol., Karolinska Inst., Stockholm, Swed.
SOURCE:                      Acta Physiologica Scandinavica (1970), 80(4), 510-18
                             CODEN: APSCAX; ISSN: 0001-6772
DOCUMENT TYPE:               Journal
LANGUAGE:                    English
GI   For diagram(s), see printed CA Issue.
AB   The cardiovascular responses of anesthetized dogs and cats to
     noradrenaline were markedly potentiated by desmethylimipramine (I)
     (0.1-1.0 mg/kg), while the responses to adrenaline and metaraminol were
     only slightly potentiated.  The pressor action of adrenaline was more
     effectively enhanced by I following β-adrenergic blockade with
     propranolol.  In analogy with the carotid occlusion response, the effect
     on peripheral vascular resistance elicited by elec. stimulation of the
     lumbar sympathetic nerves was not potentiated by I.  The reflex
     vasomotor counter-regulation was decreased by I, suggesting that
     this may be one mechanism by which I potentiates the cardiovascular
     effects of the noncatechol amine pressor agents, such as angiotensin and
     vasopressin.
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L5   ANSWER 37 OF 41     MEDLINE on STN
ACCESSION NUMBER:    70293124     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 4918143
TITLE:               Drugs and body temperature.
AUTHOR:              Lomax P
SOURCE:              International review of neurobiology, (1970) Vol. 12, pp.
                     1-43.  Ref: 202
                     Journal code: 0374740. ISSN: 0074-7742.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
                     General Review; (REVIEW)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         197011
ENTRY DATE:          Entered STN: 1 Jan 1990
                     Last Updated on STN: 1 Jan 1990
                     Entered Medline: 6 Nov 1970

L5   ANSWER 38 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN DUPLICATE 5
ACCESSION NUMBER:         1970:18834  CAPLUS
DOCUMENT NUMBER:          72:18834
TITLE:                    Potentiation by angiotensin of responses to
                          endogenously released noradrenaline in the pithed rat
AUTHOR(S):                Day, Michael D.; Owen, D. A. A.
CORPORATE SOURCE:         Dep. Pharm., Univ. Aston, Birmingham, UK
SOURCE:                   Archives Internationales de Pharmacodynamie et de
                          Therapie (1969), 179(2), 469-79
                          CODEN: AIPTAK; ISSN: 0003-9780
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   Angiotensinamide (25-300 ng/kg/min) increased pressor responses to tyramin
     e-HCl (20 µg, i.v.), tetramethylammonium chloride (TMA) (15 µg, i.v
     .), dexamphetamine sulfate, and elec. stimulation of the spinal
     sympathetic outflow in pithed rats.  These effects of angiotensin were not
     altered by desipramine pretreatment, but were decreased by
     adrenalectomy.  Noradrenaline-HCl infusion increased the pressor responses
     to tyramine and to sympathetic stimulation but to a lesser extent than did
     equipressor amts. of angiotensin.  This effect of noradrenaline was absent
     in desipramine (5 mg/kg) pretreated animals.  Tyramine (16-128
     µg/kg/min) infusions decreased the pressor responses to both
     noradrenaline and sympathetic stimulation during the infusion, but after
     the infusion both responses were increased as compared to controls.
     Desipramine pretreatment prevented both of these actions of
     tyramine.  Angiotensin apparently facilitates the release of noradrenaline
     from sympathetic vasomotor nerve endings.

L5   ANSWER 39 OF 41  CAPLUS  COPYRIGHT 2007 ACS on STN DUPLICATE 6
ACCESSION NUMBER:         1969:489830  CAPLUS
DOCUMENT NUMBER:          71:89830
TITLE:                    5-HT[5-hydroxytryptamine]autopotentiation and drug
                          induced changes in the 5-HT vasomotor
                          response in the pithed rat
AUTHOR(S):                Leach, George D. H.; Fozard, J. R.
CORPORATE SOURCE:         Sch. Stud. Pharmacol., Univ. Bradford, Bradford, UK
SOURCE:                   European Journal of Pharmacology (1969), 7(1), 14-22
                          CODEN: EJPHAZ; ISSN: 0014-2999
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   In the pithed rat, blood-pressor responses to repeated i.v. injections of
     5-hydroxytryptamine creatinine sulfate (5-HT) (4 µg. base/kg.)
     increased in size, a phenomenon designated as autopotentiation.  This
     autopotentiation was independent of mediation by catechol amines and the
     frequency of 5-HT administration but was proportional to the time elapsing
     from the beginning of an experiment  I.v. injections (mg. base/kg.) of
     cocaine-HCl (1), bretylium tosylate (20), or pheniprazine-HCl (10)
     augmented hypertensive responses to 5-HT in reserpinized, pithed rats,
     whereas amphetamine sulfate (1) and guanethidine sulfate (10) did not;
     desipramine-HCl (0.1) antagonized responses to 5-HT.  All the
     modifying drugs used potentiated pressor responses to noradrenaline
     bitartrate (150 mg. base/kg.).  Potentiation of responses to 5HT by
     cocaine-HCl and bretylium tosylate persisted after pretreatment with
     desipramine-HCl (1 mg./kg.); however, pheniprazine-HCl did not
     potentiate pressor responses to either 5-HT or noradrenaline bitartrate
     after desipramine-HCl (1 mg./kg.) administration.  Bretylium
     tosylate did not further potentiate responses to 5-HT or noradrenaline
     bitartrate after prior injection of cocaine-HCl (10 mg./kg.).  The
     relations between 5-HT autopotentiation and established catechol amine
     inactivation mechanisms are discussed.

L5   ANSWER 40 OF 41     MEDLINE on STN
ACCESSION NUMBER:    65059456     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 14254954
TITLE:               [THE ACTION OF 2 ANTIDEPRESSORS, IMIPRAMINE AND
                     DESMETHYLIMIPRAMINE, ON THE HYPOTHALAMIC AND BULBAR
                     VASOMOTOR CENTERS].
                     ACTION DE DEUX ANTID'EPRESSEURS, L'IMIPRAMINE ET LA
                     DESM'ETHYLIMIPRAMINE, SUR LES CENTRES VASOMOTEURS
                     HYPOTHALAMIQUES ET BULBAIRES.
AUTHOR:              ESPLUGUES J; SCHMITT H; SCHMITT H
SOURCE:              Comptes rendus hebdomadaires des seances de l'Academie des
                     sciences, (1964 Dec 21) Vol. 259, pp. 4876-8.
                     Journal code: 7501108. ISSN: 0001-4036.
PUB. COUNTRY:        France
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            French
FILE SEGMENT:        OLDMEDLINE; NONMEDLINE
ENTRY MONTH:         199612
ENTRY DATE:          Entered STN: 16 Jul 1999
                     Last Updated on STN: 16 Jul 1999
                     Entered Medline: 1 Dec 1996

L5   ANSWER 41 OF 41     MEDLINE on STN
ACCESSION NUMBER:    65032545     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 14228134
TITLE:               A COMPARISON OF IMIPRAMINE, CHLORPROMAZINE AND RELATED
                     DRUGS IN VARIOUS TESTS INVOLVING AUTONOMIC FUNCTIONS AND
                     ANTAGONISM OF RESERPINE.
AUTHOR:              HALLIWELL G; QUINTON R M; WILLIAMS F E
SOURCE:              British journal of pharmacology and chemotherapy, (1964
                     Oct) Vol. 23, pp. 330-50.
                     Journal code: 0154627. ISSN: 0366-0826.
PUB. COUNTRY:        ENGLAND: United Kingdom
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        OLDMEDLINE; NONMEDLINE
ENTRY MONTH:         199612
ENTRY DATE:          Entered STN: 16 Jul 1999
                     Last Updated on STN: 16 Jul 1999
                     Entered Medline: 1 Dec 1996


=> s norpramin or pertofrane
L12        867 NORPRAMIN OR PERTOFRANE

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'2003' NOT A VALID FIELD CODE
'2003' NOT A VALID FIELD CODE
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L13         765 L12 AND (AY<2003 OR PY<2003 OR PRY<2003)

=> dup rem L13
PROCESSING COMPLETED FOR L13
L14          741 DUP REM L13 (24 DUPLICATES REMOVED)

=> s vasomotor
L15       34332 VASOMOTOR

=> s L14 and L15
L16            3 L14 AND L15

=> d 1-3 L16 ibib abs

L16  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2005:369266  CAPLUS
DOCUMENT NUMBER:          142:404276
TITLE:                    Method using adrenergic α2B antagonists, alone
                          or in combination with norepinephrine reuptake
                          inhibitors or dual norepinephrine/serotonin reuptake
                          inhibitors for treating vasomotor symptoms
INVENTOR(S):              Deecher, Darlene Coleman; Beyer, Chad Edward;
                          Leventhal, Liza
PATENT ASSIGNEE(S):       Wyeth, John, and Brother Ltd., USA
SOURCE:                   PCT Int. Appl., 48 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   3
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
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     WO 2005037260        A2    20050428    WO 2004-US33754         20041013
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             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
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             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     US 2004152710        A1    20040805    US 2003-685812          20031014 <--
     WO 2004035058        A1    20040429    WO 2003-US32759         20031015 <--
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
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     US 2005130987        A1    20050616    US 2004-962897          20041012
     AU 2004281750        A1    20050428    AU 2004-281750          20041013
     CA 2539757           A1    20050428    CA 2004-2539757         20041013
     BR 2004015280         A     20061219   BR 2004-15280          20041013
PRIORITY APPLN. INFO.:                      US 2003-510897P     P  20031014
                                            US 2003-685812      A  20031014
                                            WO 2003-US32759     A  20031015
                                            US 2004-962897      A  20041012
                                            US 2002-418591P     P  20021015 <--
                                            WO 2004-US33754     W  20041013
AB   The invention discloses selective adrenergic α2B antagonists alone,
     selective adrenergic α2B antagonists in combination with
     norepinephrine reuptake inhibitors (NRI) (as a single compound or as a
     combination of two or more compds.), or selective adrenergicα2B
     antagonists in combination with dual norepinephrine reuptake
     inhibitors/serotonin reuptake inhibitors (NRI/SRI) (as a single compound or
     as a combination of two or more compds.) and methods of their use in the
     treatment of vasomotor symptoms.

L16  ANSWER 2 OF 3  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2004:354797  CAPLUS
DOCUMENT NUMBER:          140:350606
TITLE:                    Use of norepinephrine reuptake modulators for
                          preventing and treating vasomotor symptoms
INVENTOR(S):              Deecher, Darlene Coleman; Merchenthaler, Istvan
                          Joseph; Leventhal, Liza; Sipe, Kimberly Jean;
                          O'Connor, Lawrence Thomas
PATENT ASSIGNEE(S):       Wyeth, John, and Brother Ltd., USA
SOURCE:                   PCT Int. Appl., 65 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   3
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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     WO 2004035058        A1    20040429    WO 2003-US32759        20031015 <--
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             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
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     US 2004152710        A1    20040805    US 2003-685812         20031014 <--
     CA 2502032           A1    20040429    CA 2003-2502032        20031015 <--
     AU 2003282861        A1    20040504    AU 2003-282861         20031015 <--
     EP 1551413           A1    20050713    EP 2003-774853         20031015 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
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     BR 2003015355        A     20050823    BR 2003-15355          20031015 <--
     CN 1705484           A     20051207    CN 2003-80101535       20031015 <--
     JP 2006506372        T     20060223    JP 2004-545349         20031015 <--
     US 2005130987        A1    20050616    US 2004-962897         20041012
     AU 2004281750        A1    20050428    AU 2004-281750         20041013
     CA 2539757           A1    20050428    CA 2004-2539757        20041013
     WO 2005037260        A2    20050428    WO 2004-US33754        20041013
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             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
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              SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
              SN, TD, TG
     BR 2004015280          A      20061219    BR 2004-15280          20041013
PRIORITY APPLN. INFO.:                         US 2002-418591P     P  20021015 <--
                                               US 2003-685812      A  20031014
                                               US 2003-510897P     P  20031014
                                               WO 2003-US32759     W  20031015
                                               US 2004-962897      A  20041012
                                               WO 2004-US33754     W  20041013
AB   The invention discloses the use of compds. and composition of compds. that
     modulate norepinephrine levels for the prevention and treatment of
     vasomotor symptoms, such as hot flush, caused by, inter alia,
     thermoregulatory dysfunctions.  Compds. of the invention include e.g.
     desipramine.
REFERENCE COUNT:          5      THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS
                                 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16  ANSWER 3 OF 3  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2004:354778  CAPLUS
DOCUMENT NUMBER:          140:350603
TITLE:                    A method of treating vasomotor symptoms
                          using a compound having norepinephrine reuptake
                          inhibitor activity and 5-HT2a antagonistic activity
INVENTOR(S):              Deecher, Darlene Coleman; Merchenthaler, Istvan Joseph
PATENT ASSIGNEE(S):       Wyeth, John, and Brother Ltd., USA
SOURCE:                   PCT Int. Appl., 34 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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     WO 2004035036        A1    20040429    WO 2003-US32554        20031015 <--
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             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
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             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2004180879        A1    20040916    US 2003-685974         20031014 <--
     CA 2502027           A1    20040429    CA 2003-2502027        20031015 <--
     AU 2003282830        A1    20040504    AU 2003-282830         20031015 <--
     EP 1551380           A1    20050713    EP 2003-774828         20031015 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
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     BR 2003015346        A     20050823    BR 2003-15346          20031015 <--
     CN 1705475           A     20051207    CN 2003-80101558       20031015 <--
     JP 2006516023        T     20060615    JP 2004-545270         20031015 <--
PRIORITY APPLN. INFO.:                      US 2002-418516P     P  20021015 <--
                                            US 2003-685974      A  20031014
                                            WO 2003-US32554     W  20031015
AB   The invention discloses the use of compds. and compns. of compds. that
     modulate norepinephrine levels for the treatment of vasomotor
     symptoms, e.g. thermoregulatory disorders.  The invention also discloses
     the use of compds. and compns. of compds. having norepinephrine reuptake
     inhibitor (NRI) activity alone or norepinephrine reuptake inhibitor and
     serotonin reuptake inhibitor (NRI/SRI) dual activity in combination with
     5-HT2a receptor antagonist activity.
REFERENCE COUNT:         6     THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> s L2 or NRI
L17       3581 L2 OR NRI

=> s L14 and L17
L18         15 L14 AND L17

=> s L18 and (AY<2003 or PY<2003 or PRY<2003)
'2003' NOT A VALID FIELD CODE
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'2003' NOT A VALID FIELD CODE
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L19         15 L18 AND (AY<2003 OR PY<2003 OR PRY<2003)

=> d 1-5 ibib abs

L19  ANSWER 1 OF 15  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2005:369266  CAPLUS
DOCUMENT NUMBER:         142:404276
TITLE:                   Method using adrenergic α2B antagonists, alone
                         or in combination with norepinephrine
                         reuptake inhibitors or dual
                         norepinephrine/serotonin reuptake inhibitors for
                         treating vasomotor symptoms
INVENTOR(S):             Deecher, Darlene Coleman; Beyer, Chad Edward;
                         Leventhal, Liza
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 48 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  3
PATENT INFORMATION:

     PATENT NO.          KIND   DATE         APPLICATION NO.        DATE
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             SN, TD, TG
     US 2004152710        A1    20040805     US 2003-685812         20031014 <--
     WO 2004035058        A1    20040429     WO 2003-US32759        20031015 <--
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            KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
            FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
            BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2005130987          A1    20050616   US 2004-962897         20041012
     AU 2004281750          A1    20050428   AU 2004-281750         20041013
     CA 2539757             A1    20050428   CA 2004-2539757        20041013
     BR 2004015280          A     20061219   BR 2004-15280          20041013
PRIORITY APPLN. INFO.:                       US 2003-510897P     P  20031014
                                             US 2003-685812      A  20031014
                                             WO 2003-US32759     A  20031015
                                             US 2004-962897      A  20041012
                                             US 2002-418591P     P  20021015 <--
                                             WO 2004-US33754     W  20041013
AB   The invention discloses selective adrenergic α2B antagonists alone,
     selective adrenergic α2B antagonists in combination with
     norepinephrine reuptake inhibitors (NRI) (as a
     single compound or as a combination of two or more compds.), or selective
     adrenergicα2B antagonists in combination with dual
     norepinephrine reuptake inhibitors/serotonin reuptake
     inhibitors (NRI/SRI) (as a single compound or as a combination of
     two or more compds.) and methods of their use in the treatment of
     vasomotor symptoms.

L19  ANSWER 2 OF 15  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2004:354797  CAPLUS
DOCUMENT NUMBER:          140:350606
TITLE:                    Use of norepinephrine reuptake
                          modulators for preventing and treating vasomotor
                          symptoms
INVENTOR(S):              Deecher, Darlene Coleman; Merchenthaler, Istvan
                          Joseph; Leventhal, Liza; Sipe, Kimberly Jean;
                          O'Connor, Lawrence Thomas
PATENT ASSIGNEE(S):       Wyeth, John, and Brother Ltd., USA
SOURCE:                   PCT Int. Appl., 65 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   3
PATENT INFORMATION:

     PATENT NO.             KIND   DATE       APPLICATION NO.         DATE
     ---------------        ----   --------   --------------------    --------
     WO 2004035058          A1     20040429   WO 2003-US32759         20031015 <--
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE,
             GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
             LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2004152710          A1     20040805   US 2003-685812          20031014 <--
     CA 2502032             A1     20040429   CA 2003-2502032         20031015 <--
     AU 2003282861          A1     20040504   AU 2003-282861          20031015 <--
     EP 1551413             A1     20050713   EP 2003-774853          20031015 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     BR 2003015355          A      20050823   BR 2003-15355           20031015 <--
     CN 1705484             A      20051207   CN 2003-80101535        20031015 <--
     JP 2006506372          T      20060223   JP 2004-545349          20031015 <--
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          US 2005130987        A1     20050616    US 2004-962897         20041012
          AU 2004281750        A1     20050428    AU 2004-281750         20041013
          CA 2539757           A1     20050428    CA 2004-2539757        20041013
          WO 2005037260        A2     20050428    WO 2004-US33754        20041013
              W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
                  CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
                  GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
                  LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
                  NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
                  TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
              RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
                  AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
                  EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
                  SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
                  SN, TD, TG
          BR 2004015280        A      20061219    BR 2004-15280          20041013
PRIORITY APPLN. INFO.:                            US 2002-418591P     P  20021015 <--
                                                  US 2003-685812      A  20031014
                                                  US 2003-510897P     P  20031014
                                                  WO 2003-US32759     W  20031015
                                                  US 2004-962897      A  20041012
                                                  WO 2004-US33754     W  20041013
AB    The invention discloses the use of compds. and composition of compds. that
      modulate norepinephrine levels for the prevention and treatment of
      vasomotor symptoms, such as hot flush, caused by, inter alia,
      thermoregulatory dysfunctions.  Compds. of the invention include e.g.
      desipramine.
REFERENCE COUNT:         5     THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19  ANSWER 3 OF 15  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2004:354778  CAPLUS
DOCUMENT NUMBER:         140:350603
TITLE:                   A method of treating vasomotor symptoms using a
                         compound having norepinephrine
                         reuptake inhibitor activity and 5-HT2a
                         antagonistic activity
INVENTOR(S):             Deecher, Darlene Coleman; Merchenthaler, Istvan Joseph
PATENT ASSIGNEE(S):      Wyeth, John, and Brother Ltd., USA
SOURCE:                  PCT Int. Appl., 34 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

          PATENT NO.           KIND   DATE        APPLICATION NO.        DATE
          ---------------      ----   --------    --------------------   --------
          WO 2004035036        A1     20040429    WO 2003-US32554        20031015 <--
              W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
                  CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE,
                  GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
                  LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM,
                  PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
                  TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
              RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
                  KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
                  FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
                  BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
          US 2004180879        A1     20040916    US 2003-685974         20031014 <--
          CA 2502027           A1     20040429    CA 2003-2502027        20031015 <--
          AU 2003282830        A1     20040504    AU 2003-282830         20031015 <--
          EP 1551380           A1     20050713    EP 2003-774828         20031015 <--
              R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
          IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     BR 2003015346        A     20050823   BR 2003-15346         20031015 <--
     CN 1705475           A     20051207   CN 2003-80101558      20031015 <--
     JP 2006516023        T     20060615   JP 2004-545270        20031015 <--
PRIORITY APPLN. INFO.:                     US 2002-418516P    P  20021015 <--
                                           US 2003-685974     A  20031014
                                           WO 2003-US32554    W  20031015
AB   The invention discloses the use of compds. and compns. of compds. that
     modulate norepinephrine levels for the treatment of vasomotor symptoms,
     e.g. thermoregulatory disorders.  The invention also discloses the use of
     compds. and compns. of compds. having norepinephrine
     reuptake inhibitor (NRI) activity alone or
     norepinephrine reuptake inhibitor and serotonin reuptake
     inhibitor (NRI/SRI) dual activity in combination with 5-HT2a
     receptor antagonist activity.
REFERENCE COUNT:         6     THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19  ANSWER 4 OF 15  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2002049432  EMBASE
TITLE:               Reboxetine, a selective norepinephrine
                     reuptake inhibitor, is an effective and
                     well-tolerated treatment for panic disorder.
AUTHOR:              Versiani M.; Cassano G.; Perugi G.; Benedetti A.; Mastalli
                     L.; Nardi A.; Savino M.
CORPORATE SOURCE:    Prof. M. Versiani, Institute of Psychiatry, Federal
                     University of Rio de Janeiro, Rua Visconde de Piraja 407 s
                     805, Rio de Janeiro 22410-003, Brazil
SOURCE:              Journal of Clinical Psychiatry, (2002) Vol. 63, No. 1, pp.
                     31-37. .
                     Refs: 46
                     ISSN: 0160-6689  CODEN: JCLPDE
COUNTRY:             United States
DOCUMENT TYPE:       Journal; Article
FILE SEGMENT:        032     Psychiatry
                     037     Drug Literature Index
                     038     Adverse Reactions Titles
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 14 Feb 2002
                     Last Updated on STN: 14 Feb 2002
AB   Background: Tricyclic antidepressants and selective serotonin reuptake
     inhibitors (SSRIs) as well as benzodiazepines have been shown to be
     effective for the treatment of panic disorder.  The introduction of SSRIs
     has enabled a greater understanding of the role of serotonin in the
     etiology of panic disorder; however, the role of norepinephrine has been
     more challenging to ascertain.  The aim of this study was to determine the
     efficacy and tolerability of reboxetine, a novel selective
     norepinephrine reuptake inhibitor, in patients with
     panic disorder with and without agoraphobia.  Method: Eighty-two patients
     (aged 18-65 years) with DSM-III-R panic disorder, with or without
     agoraphobia, were randomly assigned to receive 6 to 8 mg/day of reboxetine
     (42 patients) or placebo (40 patients) for 8 weeks in this
     placebo-controlled, parallel-group, double-blind clinical trial.  Results:
     Of the 82 patients enrolled in the trial, 75 were considered in the
     analysis (37 patients in the reboxetine group and 38 patients in the
     placebo group).  At last assessment, there was a significant reduction in
     the mean number of panic attacks (range, 9.3-1.2) and phobic symptoms
     (range, 8.1-3.2) in the reboxetine group compared with the placebo group
     (ranges, 8.5-5.8 and 7.7-5.2, respectively; p < .05).  Improvement in
     Hamilton Rating Scale for Depression, Hopkins Symptom Checklist-90, and
     Sheehan Disability Scale scores were also greater in the reboxetine group
     compared with the placebo group.  Adverse events reported more frequently
     with reboxetine than placebo included dry mouth (36% vs. 16%),
     constipation (27% vs. 22%), and insomnia (26% vs. 22%). Conclusion:
     Reboxetine was effective and well tolerated in the treatment of panic
     disorder.

L19  ANSWER 5 OF 15  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2001085671  EMBASE
TITLE:               Possible neurobiological mechanisms underlying faster onset
                     of antidepressant action.
AUTHOR:              Blier P.
CORPORATE SOURCE:    Dr. P. Blier, Department of Psychiatry, Brain Institute,
                     University of Florida, 100 Newell Dr., Gainesville, FL
                     32611-0256, United States
SOURCE:              Journal of Clinical Psychiatry, (2001) Vol. 62, No. SUPPL.
                     4, pp. 7-11. .
                     Refs: 46
                     ISSN: 0160-6689  CODEN: JCLPDE
COUNTRY:             United States
DOCUMENT TYPE:       Journal; Article
FILE SEGMENT:        030     Pharmacology
                     032     Psychiatry
                     037     Drug Literature Index
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 16 Mar 2001
                     Last Updated on STN: 16 Mar 2001
AB   All antidepressant drugs have a delayed onset of action.  There is,
     however, evidence that some agents may attenuate depressive symptoms more
     rapidly than others.  The present review examines the mechanisms by which
     selective serotonin reuptake inhibitors, the dual 5-HT
     norepinephrine reuptake inhibitor venlafaxine, and the
     α(2)-adrenoceptor antagonist mirtazapine alter 5-HT and/or
     norepinephrine neurotransmission.  Particular attention is given to the
     time course with which these effects manifest themselves in relation to
     the possibility that these 3 types of drugs may act more rapidly, or exert
     a greater antidepressant action, than other agents.  Based on the effects
     of antidepressant drugs presently available, strategies to accelerate or
     augment the antidepressant response are described, some of which have
     already been examined in patients.


=>  L19

L19 IS NOT A RECOGNIZED COMMAND
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"HELP COMMANDS" at an arrow prompt (=>).

=> d L19 6-15 ibib abs

L19  ANSWER 6 OF 15  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2000265457  EMBASE
TITLE:               Clinical efficacy of reboxetine in major depression.
AUTHOR:              Schatzberg A.F.
CORPORATE SOURCE:    Dr. A.F. Schatzberg, Dept. of Psychiatry/Behavioral Sci.,
                     Stanford Univ. School of Medicine, Stanford, CA 94305-5717,
                     United States
SOURCE:              Journal of Clinical Psychiatry, (2000) Vol. 61, No. SUPPL.
                     10, pp. 31-38. .
                     Refs: 38
                     ISSN: 0160-6689  CODEN: JCLPDE
COUNTRY:             United States
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DOCUMENT TYPE:       Journal; General Review
FILE SEGMENT:        030     Pharmacology
                     032     Psychiatry
                     037     Drug Literature Index
                     038     Adverse Reactions Titles
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 17 Aug 2000
                     Last Updated on STN: 17 Aug 2000
AB   The past decade has witnessed the advent of selective serotonin reuptake
     inhibitors (SSRIs) as first-line treatments for major depression.  Still,
     there is considerable debate as to whether these agents are as effective
     or as potent as the first- generation tricyclic antidepressants (TCAs) or
     the mixed reuptake inhibitor, venlafaxine, all of which exert considerable
     effect on norepinephrine (NE) reuptake.  Recently, reboxetine, a selective
     NE reuptake inhibitor (selective NRI), has been introduced in
     Europe.  This drug has only a minimal affinity for muscarinic
     acetylcholine receptors and therefore causes less dry mouth, constipation,
     or other such effects than do the TCAs.  Reboxetine does not block
     serotonin reuptake or α1 receptors and, thus, does not appear to
     produce significant nausea, diarrhea, or hypotension.  Unlike other
     antidepressants, reboxetine appears to be nonsedating.  Data on acute and
     long-term clinical efficacy and safety from double-blind,
     placebo-controlled, and active comparator studies with reboxetine are
     reviewed.  These studies indicate that reboxetine is significantly more
     effective than placebo and as effective as fluoxetine in reducing
     depressive symptoms.  Improvements in social adjustments were reported to
     be more favorable with reboxetine than with fluoxetine.  Further, data
     from controlled clinical trials have shown that the side effect profile
     for reboxetine is relatively benign.  The clinical implications of studies
     on reboxetine are discussed with an eye toward understanding the potential
     role NE reuptake blockers may play in the treatment of patients with major
     depression.

L19  ANSWER 7 OF 15  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2000265456  EMBASE
TITLE:               Norepinephrine involvement in antidepressant action.
AUTHOR:              Frazer A.
CORPORATE SOURCE:    Dr. A. Frazer, Department of Pharmacology, Univ. of Texas
                     Health Sci. Center, 7703 Floyd Curl Dr., San Antonio, TX
                     78284-7764, United States. frazer@uthscsa.edu
SOURCE:              Journal of Clinical Psychiatry, (2000) Vol. 61, No. SUPPL.
                     10, pp. 25-30. .
                     Refs: 44
                     ISSN: 0160-6689  CODEN: JCLPDE
COUNTRY:             United States
DOCUMENT TYPE:       Journal; General Review
FILE SEGMENT:        030     Pharmacology
                     032     Psychiatry
                     037     Drug Literature Index
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 17 Aug 2000
                     Last Updated on STN: 17 Aug 2000
AB   Because of the introduction and popularity of the selective serotonin
     reuptake inhibitor (SSRI) antidepressants, much attention was centered on
     the indolealkylamine 5- hydroxytryptamine, or serotonin.  To some extent,
     this focus on serotonin occurred at the expense of the catecholamine
     neurotransmitter norepinephrine.  Nevertheless, it has been apparent for
     almost 40 years that selective blockers of norepinephrine
     reuptake may be antidepressants (e.g., desipramine).  This brief
     review covers the acute pharmacologic effects that may be responsible for
     the efficacy of currently marketed antidepressants as well as that of
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reboxetine, a newly developed selective norepinephrine reuptake inhibitor. Also discussed is the fact that the acute pharmacologic profile of selective reuptake inhibitors often predicts effects they produce when given long term. For example, the long-term administration of SSRIs produces certain effects on serotonergic systems, but not noradrenergic ones. By contrast, selective norepinephrine reuptake inhibitors, when given long term, modify certain noradrenergic parameters, but not serotonergic indices. Finally, it is speculated how drugs that enhance central noradrenergic transmission might ameliorate the symptoms of depression.

L19 ANSWER 8 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2000161114 EMBASE
TITLE: A systematic review of newer pharmacotherapies for depression in adults: Evidence report summary.
AUTHOR: Williams J.W. Jr.; Mulrow C.D.; Chiquette E.; Noel P.H.; Aguilar C.; Cornell J.
CORPORATE SOURCE: Dr. J.W. Williams Jr., Department of Medicine, Mail Code 7879, Univ. of Texas Health Science Center, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, United States
SOURCE: Annals of Internal Medicine, (2 May 2000) Vol. 132, No. 9, pp. 743-756. .
Refs: 116
ISSN: 0003-4819 CODEN: AIMEAS
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 25 May 2000
Last Updated on STN: 25 May 2000

AB Background: Depressive disorders are persistent, recurring illnesses that cause great suffering for patients and their families. Purpose: To evaluate the benefits and adverse effects of newer pharmacotherapies and herbal treatments for depressive disorders in adults and adolescents. Data Sources: English-language and non-English-language literature from 1980 to January 1998 was identified from a specialized registry of controlled trials, meta-analyses, and experts. Study Selection: Randomized trials evaluating newer antidepressants (such as serotonin reuptake inhibitors, serotonin norepinephrine reuptake inhibitors, and St. John's wort) that reported clinical outcomes were selected. Data Extraction: Two persons independently abstracted data that were then synthesized descriptively; some data were pooled by using a random-effects model. Data Synthesis: Of 315 eligible trials, most evaluated antidepressants in adults with major depression, were conducted among outpatients, and examined acute-phase treatment. Newer antidepressants were more effective than placebo for major depression (relative benefit, 1.6 [95% CI, 1.5 to 1.7]) and dysthymia (relative benefit, 1.7 [CI, 1.3 to 2.3]). They were effective among older adults and primary care patients. Efficacy did not differ among newer agents or between newer and older agents. Hypericum (St. John's wort) was more effective than placebo for mild to moderate depression (risk ratio, 1.9 [CI, 1.2 to 2.8]), but publication bias may have inflated the estimate of benefit. Newer and older antidepressants did not differ for overall discontinuation rates, but side effect profiles varied significantly. Data were insufficient for determining the efficacy of newer antidepressants for subsyndromal depression, depression with coexisting medical or psychiatric illness, or depression in adolescents. Conclusions: Newer antidepressants are clearly effective in treating depressive disorders in diverse settings. Because of similar efficacy, both newer and older antidepressants should be considered when making treatment decisions. Better information is urgently needed on the

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     efficacy of newer antidepressants in patients with nonmajor depression and
     in special populations, including adolescents.

L19  ANSWER 9 OF 15  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2000145030  EMBASE
TITLE:               Are all antidepressants the same?.
AUTHOR:              Moller H.-J.
CORPORATE SOURCE:    Dr. H.-J. Moller, Department of Psychiatry, Ludwig
                     Maximilians University, Nussbaumstrasse 7, D-80336 Munich,
                     Germany
SOURCE:              Journal of Clinical Psychiatry, (2000) Vol. 61, No. SUPPL.
                     6, pp. 24-28. .
                     Refs: 30
                     ISSN: 0160-6689  CODEN: JCLPDE
COUNTRY:             United States
DOCUMENT TYPE:       Journal; Conference Article
FILE SEGMENT:        030      Pharmacology
                     032      Psychiatry
                     037      Drug Literature Index
                     038      Adverse Reactions Titles
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 11 May 2000
                     Last Updated on STN: 11 May 2000
AB   Historically, the clinician's choice of antidepressant agent has been
     determined largely by consideration of tolerability, given the perception
     that the therapeutic efficacy of the various antidepressants was broadly
     comparable.  However, with the advent of the newer, more selective
     antidepressants, indications of variation in clinical efficacy have begun
     to emerge.  The advent of the selective serotonin reuptake inhibitors has
     been welcomed by patients, largely owing to their superior tolerability
     profile compared with older antidepressants.  However, severely depressed
     patients appear to benefit particularly from agents that include a
     noradrenergic mode of action, such as tricyclic antidepressants and the
     modem dual-action antidepressants mirtazapine, venlafaxine (at higher
     doses), and milnacipran.  In addition, a noradrenergic component may offer
     superior efficacy in social functioning.  The recent development of a
     novel, selective norepinephrine reuptake inhibitor
     (reboxetine) with proven efficacy in a range of depressed patients will
     permit the investigation of the relevance of the noradrenergic approach.
     Clinical observations of the effects of the newer, more selective
     antidepressants are important in our understanding of precise mode of
     action, variation in efficacy and tolerability, and comparative usefulness
     in clinical practice.

L19  ANSWER 10 OF 15  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2000084121  EMBASE
TITLE:               New approaches to the treatment of refractory depression.
AUTHOR:              Fava M.
CORPORATE SOURCE:    Dr. M. Fava, Depression Clinical/Research Program,
                     Massachusetts General Hospital, WACC 815, 15 Parkman St.,
                     Boston, MA 02114, United States. mfava@partners.org
SOURCE:              Journal of Clinical Psychiatry, (2000) Vol. 61, No. SUPPL.
                     1, pp. 26-32. .
                     Refs: 75
                     ISSN: 0160-6689  CODEN: JCLPDE
COUNTRY:             United States
DOCUMENT TYPE:       Journal; General Review
FILE SEGMENT:        032      Psychiatry
                     037      Drug Literature Index
                     038      Adverse Reactions Titles
LANGUAGE:            English
```

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Mar 2000
Last Updated on STN: 16 Mar 2000

AB Although the majority of patients with depression respond well to their initial pharmacologic treatment, as many as 30% to 45% fail to achieve an adequate response. In addition to the more traditional lithium and thyroid hormone augmentation strategies, a number of new pharmacotherapeutic approaches are currently being used to help manage refractory depression, including the addition of another agent or a switch to another antidepressant. Augmentation and switching strategies are often selected in order to obtain a different neurochemical effect (e.g., adding a relatively noradrenergic agent to a relatively serotonergic antidepressant). In particular, several studies have suggested that depressed patients refractory to treatment with selective serotonin reuptake inhibitors (SSRIs) may show a good response to newer agents that have a pharmacologic profile distinct from the SSRIs. Furthermore, preliminary studies have shown that the addition of SSRIs to either noradrenergic drugs such as the tricyclic antidepressants (TCAs) or dopaminergic agents may be efficacious, even though concerns about drug-drug interactions and tricyclic cardiac toxicity have limited the use of TCA-SSRI combinations. The introduction of reboxetine, a relatively selective norepinephrine reuptake inhibitor, may increase the use of the latter therapeutic approach because of its improved safety profile compared with the TCAs. The review of treatment options for refractory depression that follows will outline the advantages, disadvantages, and level of support for a number of new treatment strategies.

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ACCESSION NUMBER: 1999434840 EMBASE
TITLE: The functional anatomy, neurochemistry, and pharmacology of anxiety.
AUTHOR: Ninan P.T.
CORPORATE SOURCE: Dr. P.T. Ninan, Emory University School of Medicine, 1841 Clifton Rd., Atlanta, GA 30329, United States
SOURCE: Journal of Clinical Psychiatry, (1999) Vol. 60, No. 12, pp. 12-17. .
Refs: 48.
ISSN: 0160-6689 CODEN: JCLPDE
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 29 Dec 1999
Last Updated on STN: 29 Dec 1999

AB The functional anatomy of anxiety involves amygdala-based neurocircuits with critical reciprocal connections to the medial prefrontal cortex. Traumatic experiences leave emotional imprints involving the amygdala, with facilitated fear-conditioned associations involving declarative memory traces. Avoidance conditioning is an additional component. An understanding of the functional anatomy of anxiety allows for a new perspective on the various anxiety disorders. The neurotransmitters involved in these circuits are reviewed for their relevance to the pharmacologic choices in the treatment of anxiety. Potent serotonin reuptake inhibitors appear to have superior efficacy in many of the anxiety disorders, with indications that norepinephrine reuptake inhibitors have an advantage in severe forms of major depression. Medications with dual effects - blocking reuptake of both serotonin and norepinephrine (e.g., clomipramine and venlafaxine XR) - have superior benefits in achieving remission in major depression and GAD.

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     These medications may also offer a faster onset of action and
     theoretically superior benefits in patients with comorbid anxiety disorder
     and major depression.

L19  ANSWER 12 OF 15  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
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ACCESSION NUMBER:    1999307995  EMBASE
TITLE:               Antidepressant discontinuation-related mania: Critical
                     prospective observation and theoretical implications in
                     bipolar disorder.
AUTHOR:              Goldstein T.R.; Frye M.A.; Denicoff K.D.; Smith-Jackson E.;
                     Leverich G.S.; Bryan A.L.; Ali S.O.; Post R.M.
CORPORATE SOURCE:    Dr. R.M. Post, National Institute of Mental Health,
                     Biological Psychiatry Branch, Building 10, Bethesda, MD
                     20892-1272, United States
SOURCE:              Journal of Clinical Psychiatry, (1999) Vol. 60, No. 8, pp.
                     563-567. .
                     Refs: 39
                     ISSN: 0160-6689  CODEN: JCLPDE
COUNTRY:             United States
DOCUMENT TYPE:       Journal; Article
FILE SEGMENT:        032     Psychiatry
                     037     Drug Literature Index
LANGUAGE:            English
SUMMARY LANGUAGE:    English
ENTRY DATE:          Entered STN: 16 Sep 1999
                     Last Updated on STN: 16 Sep 1999
AB   Background: Development of manic symptoms on antidepressant
     discontinuation has primarily been reported in unipolar patients.  This
     case series presents preliminary evidence for a similar phenomenon in
     bipolar patients.  Method: Prospectively obtained life chart ratings of 73
     bipolar patients at the National Institute of Mental Health were reviewed
     for manic episodes that emerged during antidepressant taper or
     discontinuation.  Medical records were utilized as a corroborative
     resource.  Six cases of antidepressant discontinuation-related mania were
     identified and critically evaluated.  Results: All patients were taking
     conventional mood stabilizers.  The patients were on antidepressant
     treatment a mean of 6.5 months prior to taper, which lasted an average of
     20 days (range, 1-43 days).  First manic symptoms emerged, on average, 2
     weeks into the taper (range, 1-23 days).  These 6 cases of antidepressant
     discontinuation-related mania involved 3 selective serotonin reuptake
     inhibitors (SSRIs), 2 tricyclic antidepressants (TCAs), and 1 serotonin-
     norepinephrine reuptake inhibitor.  Mean length of the
     ensuing manic episode was 27.8 days (range, 12-49 days).  Potential
     confounds such as antidepressant induction, phenomenological misdiagnosis
     of agitated depression, physiologic drug withdrawal syndrome, and course
     of illness were carefully evaluated and determined to be noncontributory.
     Conclusion: These 6 cases suggest a paradoxical effect whereby
     antidepressant discontinuation actually induces mania in spite of adequate
     concomitant moodstabilizing treatment.  These preliminary observations, if
     replicated in larger and controlled prospective studies, suggest the need
     for further consideration of the potential biochemical mechanisms involved
     so that new preventive treatment approaches can be assessed.

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     reserved on STN
ACCESSION NUMBER:    1999218635  EMBASE
TITLE:               Reboxetine, a unique selective NRI, prevents
                     relapse and recurrence in long-term treatment of major
                     depressive disorder.
AUTHOR:              Versiani M.; Mehilane L.; Gaszner P.; Arnaud-Castiglioni R.
CORPORATE SOURCE:    Dr. M. Versiani, Institute of Psychiatry, Federal
                     University, Rua Visconde de Piraja 407 s, Rio de Janeiro
                     22410-003, Brazil
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SOURCE: Journal of Clinical Psychiatry, (1999) Vol. 60, No. 6, pp. 400-406. .
Refs: 35
ISSN: 0160-6689 CODEN: JCLPDE
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 8 Jul 1999
Last Updated on STN: 8 Jul 1999

AB Background: The long-term efficacy and tolerability of the antidepressant reboxetine, a unique selective norepinephrine reuptake inhibitor (selective NRI), were assessed in an international study. Method: Two hundred eighty-three patients with recurrent DSM-III-R major depression who responded to 6 weeks of reboxetine treatment ($\geq$ 50% decrease in Hamilton Rating Scale for Depression [HAM-D] total score) were randomly assigned to receive reboxetine or placebo for 46 weeks in a double-blind phase. Relapse ($\geq$ 50% increase in HAM-D total score and/or a HAM-D total score ≥ 18) rate was the principal assessment criterion and included patients who experienced relapse or recurrence. Only patients who remained relapse-free at the end of the first 6-month treatment period were included in the relapse rate assessment at the end of the second 6-month treatment period. Results: Reboxetine was associated with a markedly lower relapse rate than placebo (22% vs. 56%; $p < .001$) and a greater cumulative probability of a maintained response ($p = .0001$) during long-term treatment. Patients in remission (HAM- D total score ≤ 10) at the time of random assignment were less likely to relapse (16% reboxetine, 48% placebo; $p < .001$). The proportion of patients who were relapse-free and therefore remained in the study was significantly ($p \leq .001$) higher among those on reboxetine treatment than on placebo at the end of the first (61% vs. 40%) and second (88% vs. 59%) 6 months of treatment. Additional efficacy measures supported these findings. The incidence of adverse events with reboxetine was low and comparable with that for placebo. Discontinuation due to adverse events occurred infrequently. Conclusion: Reboxetine treatment over 1 year is more effective than placebo in the prevention of relapse in patients with recurrent depression. The low relapse rates at the end of the second 6 months of treatment further suggest that reboxetine effectively prevents recurrence of depressive symptoms following episode resolution. Reboxetine is well tolerated in long-term treatment of depression, a finding that bodes well for long-term patient compliance.

L19 ANSWER 14 OF 15 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1999110001 EMBASE
TITLE: Treatment options for refractory depression.
AUTHOR: Shelton R.C.; Schatzberg; Nemeroff; Feighner; Evans; Gorman
CORPORATE SOURCE: Dr. R.C. Shelton, Dept. of Psychiat./Pharmacology, Vanderbilt Univ. School of Medicine, 1500 21st Ave. S., Nashville, TN 37212, United States.
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SOURCE: Journal of Clinical Psychiatry, (1999) Vol. 60, No. SUPPL. 4, pp. 57-63. .
Refs: 54
ISSN: 0160-6689 CODEN: JCLPDE
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

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ENTRY DATE:           Entered STN: 10 May 1999
                      Last Updated on STN: 10 May 1999
AB    A significant proportion of patients with depressive disorders do not
      experience a full response with antidepressant treatment.  Fortunately,
      most eventually remit, even though the time to response may be
      significantly delayed in many patients.  A variety of options exist to
      deal with these difficult clinical situations.  Established strategies
      include switching to an antidepressant of an alternative class (e.g.,
      tricyclic to a monoamine oxidase inhibitor [MAOI] or selective serotonin
      reuptake inhibitor [SSRI]), electroconvulsive therapy (ECT), and
      augmentation with lithium or thyroid hormone.  Promising alternatives
      include combined serotonin and norepinephrine enhancement strategies
      (e.g., SSRI plus serotonin norepinephrine reuptake
      inhibitor [NSRI] or higher doses of venlafaxine or fluoxetine), steroid
      suppression therapy, augmentation with atypical antipsychotics, and
      psychotherapy.

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ACCESSION NUMBER:     1999109999  EMBASE
TITLE:                SSRIS and SNRIs: Broad spectrum of efficacy beyond major
                      depression.
AUTHOR:               Gorman J.M.; Kent J.M.; Evans; Schatzberg; Nemeroff
CORPORATE SOURCE:     Dr. J.M. Gorman, New York State Psychiatric Institute, 722
                      W. 168th St., New York, NY 10032, United States
SOURCE:               Journal of Clinical Psychiatry, (1999) Vol. 60, No. SUPPL.
                      4, pp. 33-39. .
                      Refs: 35
                      ISSN: 0160-6689  CODEN: JCLPDE
COUNTRY:              United States
DOCUMENT TYPE:        Journal; General Review
FILE SEGMENT:         032     Psychiatry
                      037     Drug Literature Index
LANGUAGE:             English
SUMMARY LANGUAGE:     English
ENTRY DATE:           Entered STN: 10 May 1999
                      Last Updated on STN: 10 May 1999
AB    Originally studied and introduced for the treatment of depression, the
      selective serotonin reuptake inhibitors (SSRIs) and serotonin/
      norepinephrine reuptake inhibitors (SNRIs) have proven
      effective for a broad range of psychiatric illnesses, including several
      anxiety disorders, bulimia, and dysthymia.  These drugs have in common
      important effects on the serotonergic (5-HT) neurotransmission system,
      which is involved in mediating a substantial number of important
      functions, including mood, aggression, sexual behavior, and pain.  In
      addition, some of the new antidepressants, like venlafaxine/venlafaxine
      XR, also have effects on the noradrenergic neurotransmission system, which
      also appears important in mood and anxiety disorders.  These new drugs,
      because of their specificity for the serotonin and norepinephrine
      reuptake proteins, lack most of the adverse side effects of
      tricyclic antidepressants and monoamine oxidase inhibitors.  Consequently,
      in addition to being the usual first-line treatments for major depression,
      they are also first-line for panic disorder, obsessive-compulsive
      disorder, social phobia, posttraumatic stress disorder, and bulimia.  They
      may also be the best medication treatments for dysthymia and generalized
      anxiety disorder.  Further advances in psychopharmacology will be driven
      by discoveries from brain imaging and molecular biological research.


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